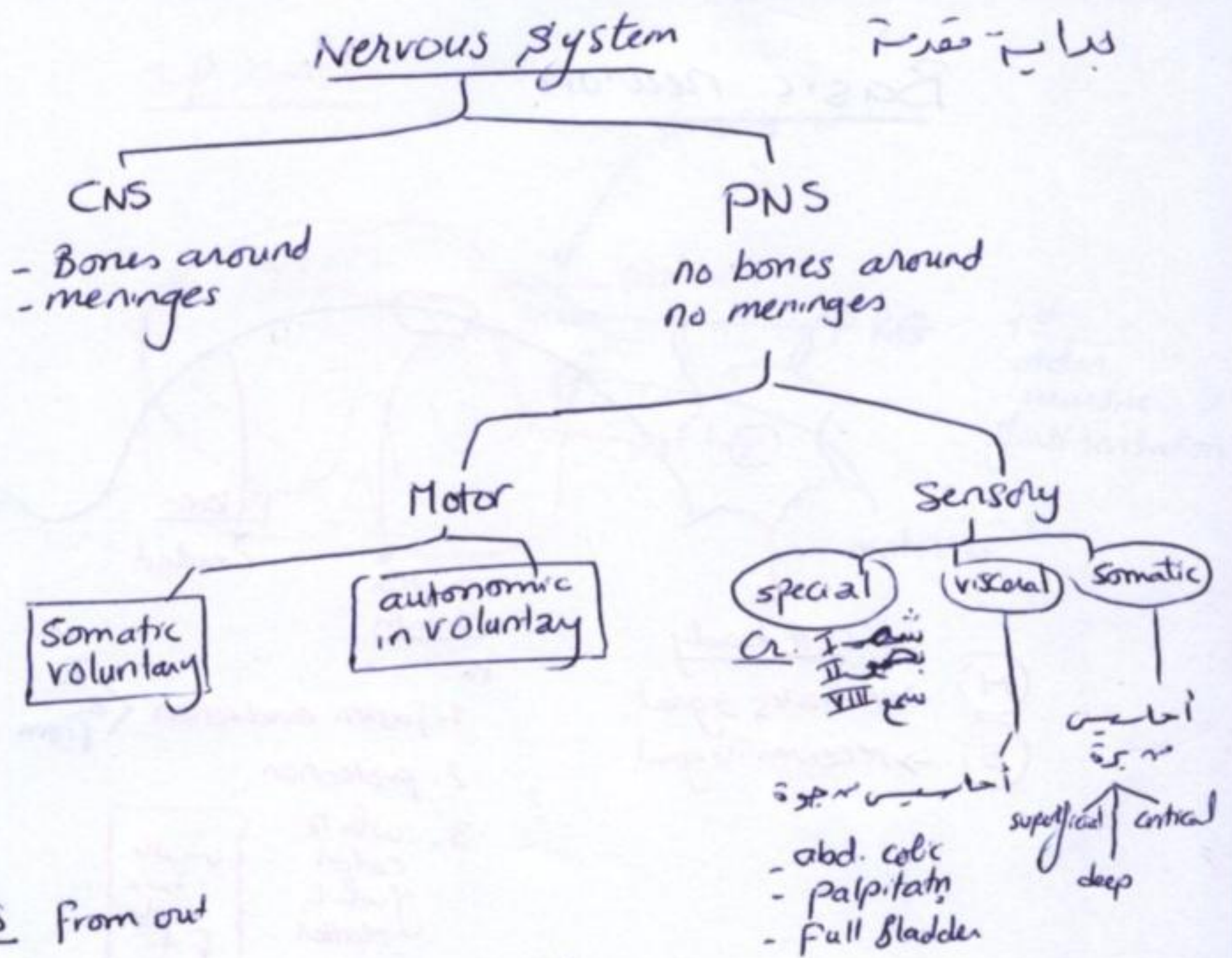
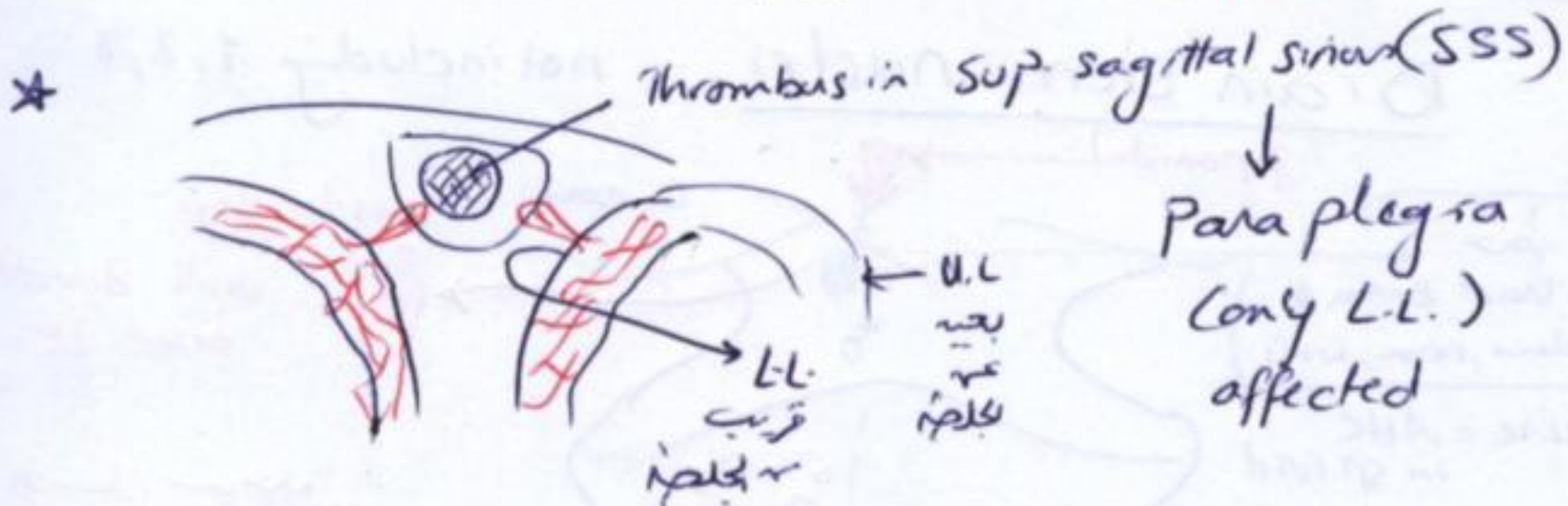
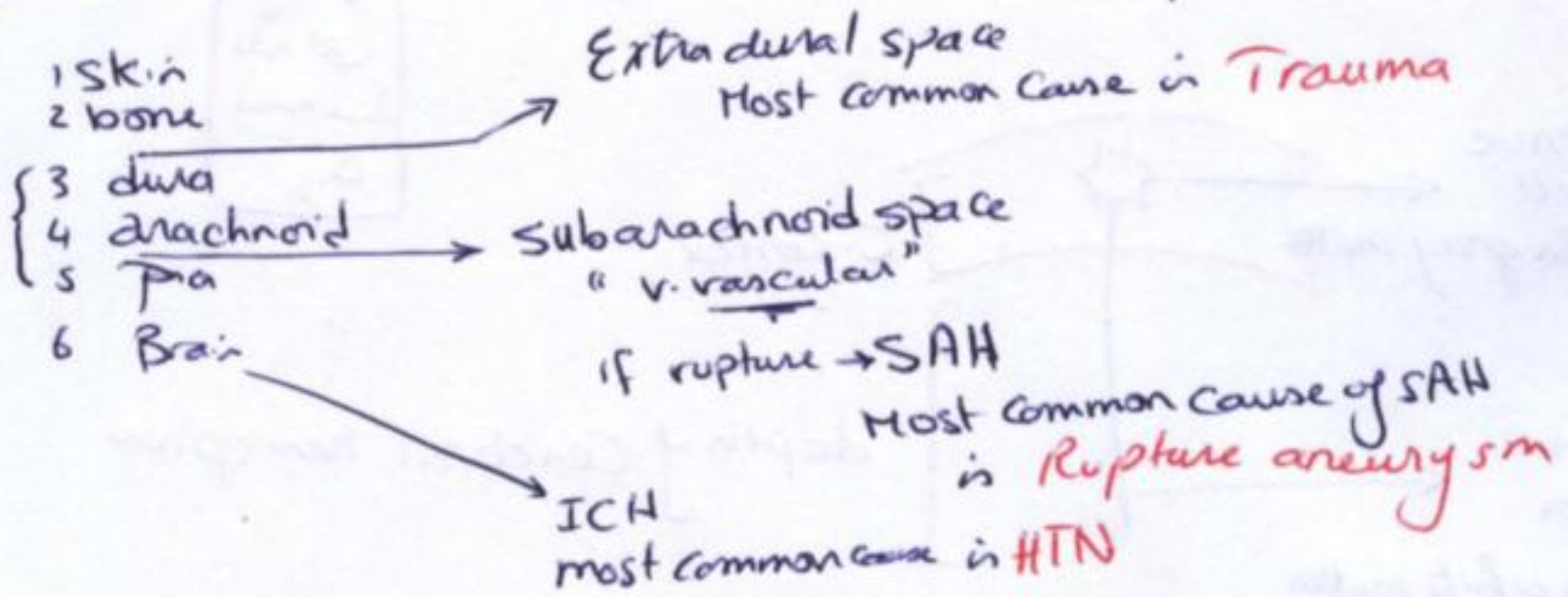




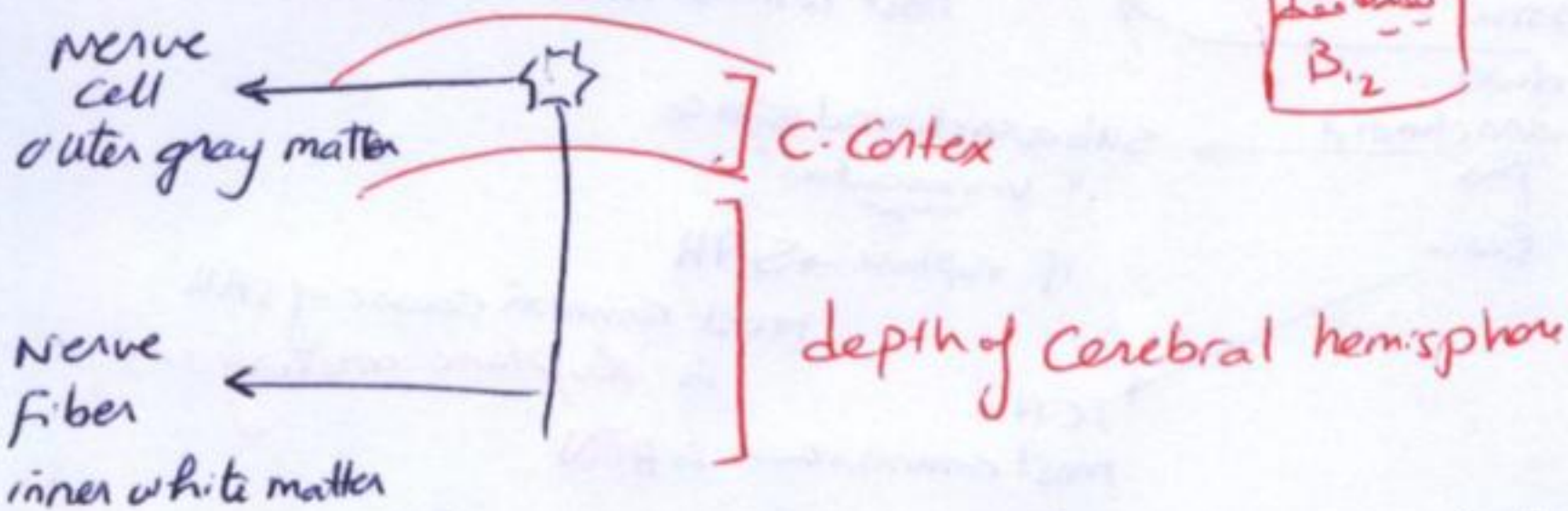
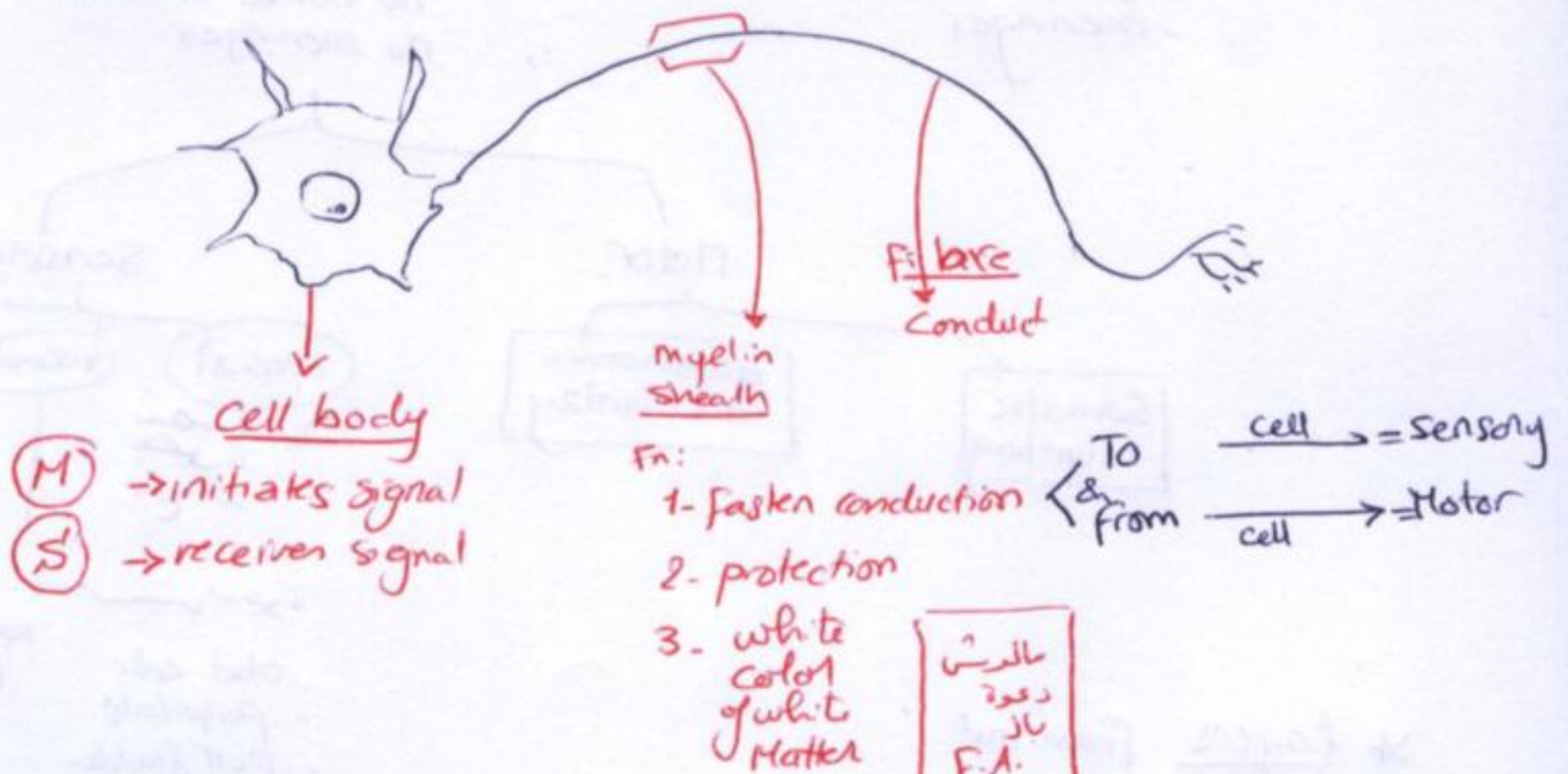
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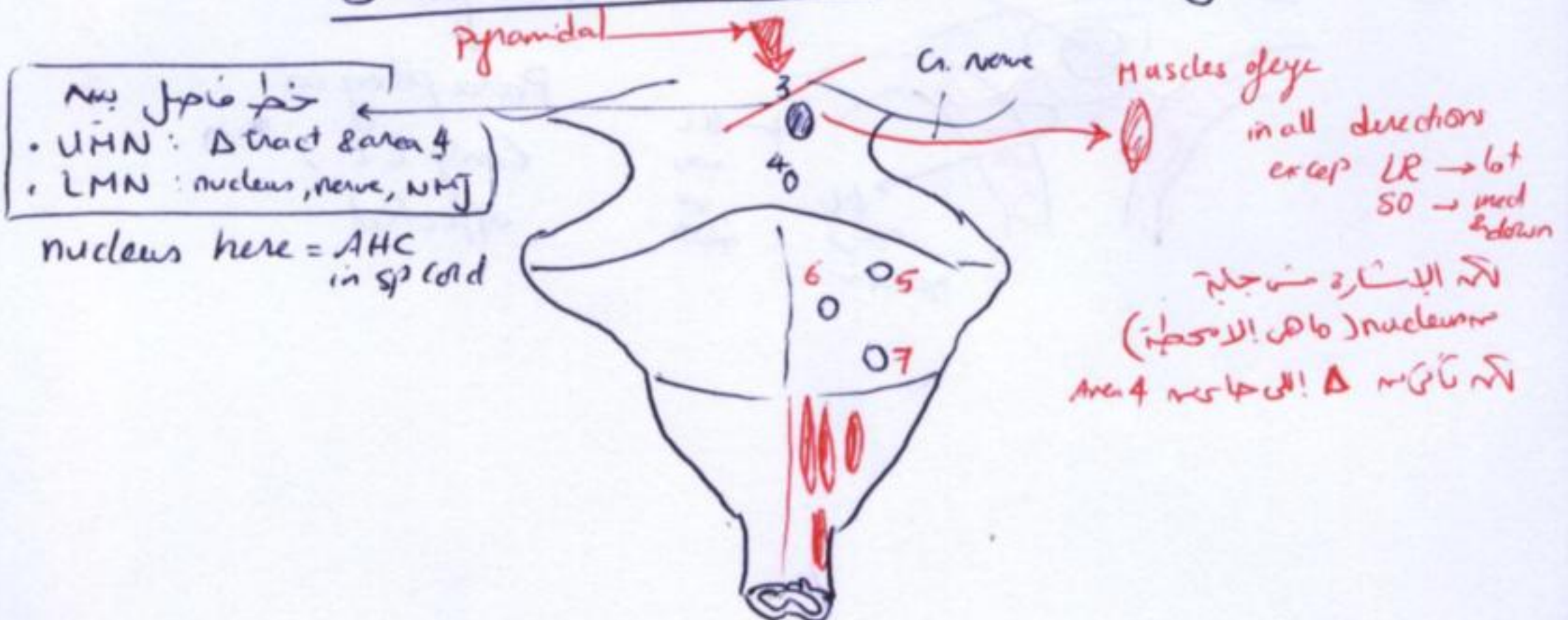
VISIT...

LANZAROTE
Caliente.COM

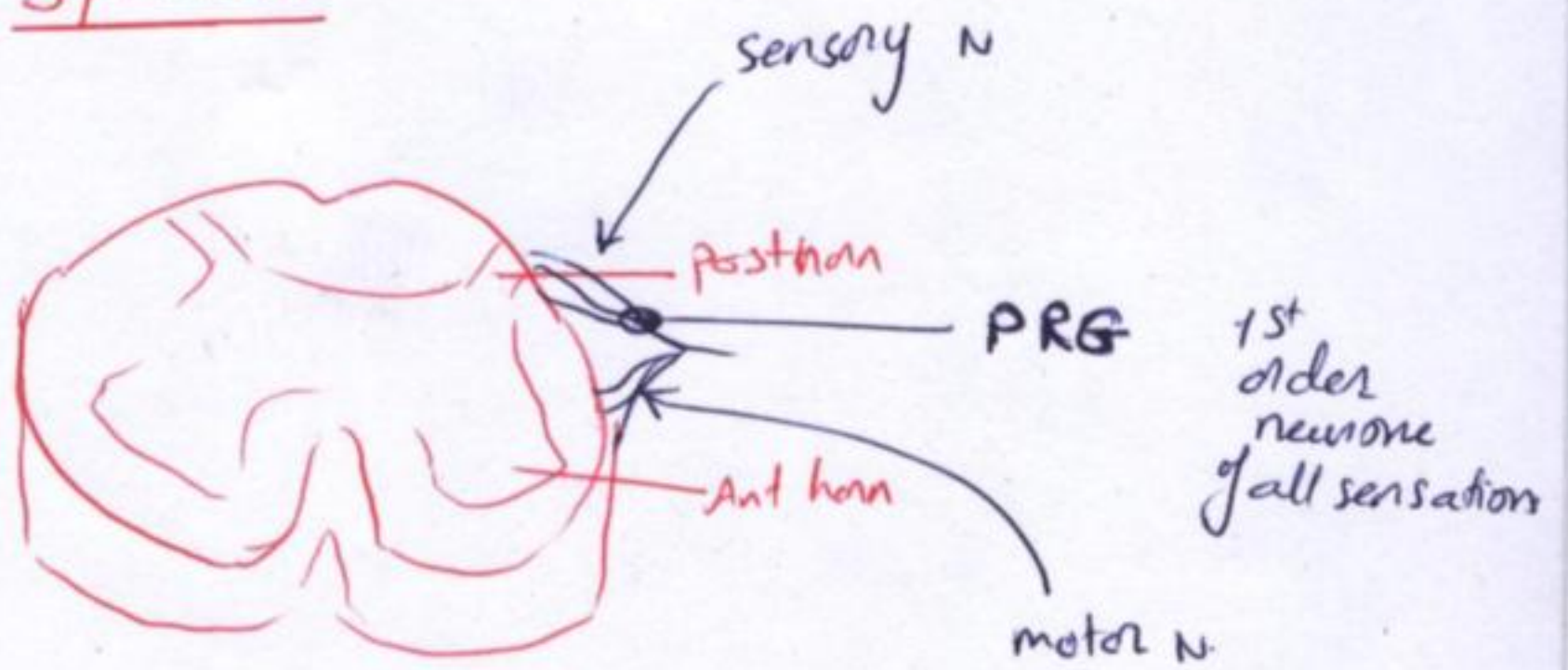
Basic neuron



Brain stem nuclei not including 1, 2, 8



Sp. cord



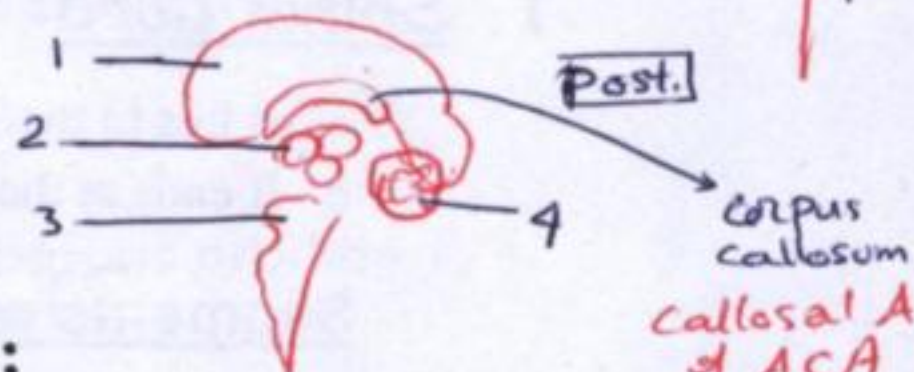
CENTRAL NERVOUS SYSTEM

The CNS is formed of 2 main parts:

I. INTRACRANIAL PART (Brain)

1. Two cerebral hemispheres.
2. Basal ganglia.
3. Brain stem.
4. Cerebellum.

Ant



1. Two cerebral hemispheres:

- CONNECTION: They are connected to each other by the CORPUS CALLOSUM.

- SURFACE: The surface of each hemisphere is divided into 4 LOBES:

1. Frontal.
2. Parietal.
3. Temporal.
4. Occipital.

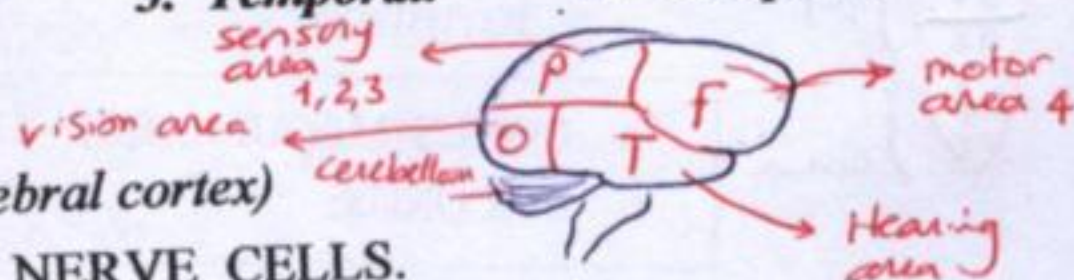
- CEREBRAL LOBES:

1. Outer gray matter:

- o It is composed of: NERVE CELLS.
- o It contains certain AREAS that control specific functions.

2. Inner white matter: (depth of the cerebral hemisphere)

- o It is composed of: NERVE FIBRES.
- o It CONDUCTS IMPULSES to & from the cerebral cortex.



2. Basal nuclei:

- At the base of each cerebral hemisphere (At various levels deep in the white matter):

- BASAL GANGLIA: Caudate, Putamen, Globus pallidus.

- THALAMUS, SUBTHALAMUS, HYPOTHALAMUS.

3. Brain stem:

"3 parts from above downwards"

- MIDBRAIN: contains the motor nuclei of the cranial nerves 3 & 4.
- PONS: contains the motor nuclei of the cranial nerves 5, 6, 7.
- MEDULLA: contains the motor nuclei of the cranial nerves 9, 10, 11, 12.

- The 1st, 2nd & 8th cranial nerves have no motor nuclei.
- They are SENSORY NERVES concerned with special sensations.

4. Cerebellum:

- It lies at the BACK & the BOTTOM of the cranium behind the brain stem.

- 1- Hormones
- 2- sleep centre, satiety centre

II. SPINAL PART

- canal هو في
canal تحت في
1. Spinal Cord.
 2. Cauda Equina. *canal من*

- From sacrum to L₁ in canal
- width of thumb
- 2 way communication

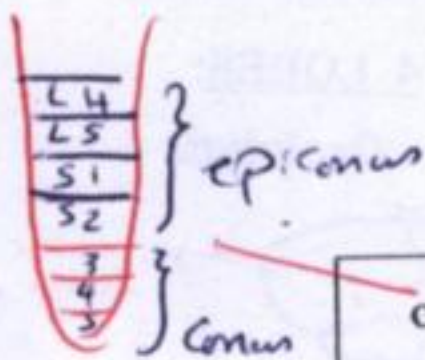
1. Spinal Cord:

- o It lies in the spinal canal.
- o It ends at the lower border of the 1st lumbar vertebra (L1).

Segments of the spinal cord:

- Cervical segments: 8 segments.
- Thoracic segments: 12 segments.
- Lumbar segments: 5 segments.
- Sacral segments: 5 segments.

الوحيد
للنارية
من
عظم
(7)



- o CONUS MEDULLARIS: The lowermost 3 segments of the spinal cord (S3, 4, 5).
- o EPICONUS: The 4 segments above the conus medullaris (L4, 5, S1, 2).

Section in the spinal cord:

عكس تقسيم المخ
التي برة
Gray
white

- It contains: gray matter (cells) surrounded by white matter (fibres).
- The GRAY MATTER: H-shaped in a transverse section & is formed of:
 - 2 anterior horns: MOTOR function.
 - 2 posterior horns: SENSORY function.
- The WHITE MATTER: contains nerve fibres arranged into tracts:

1. The important ascending tracts are:

- Lateral & ventral spinothalamic: for superficial sensations.
- Posterior column: for deep sensations.
- Spinocerebellar: for cerebellar information.

→ feed back
عنه اتزانك

2. The important descending tracts are:

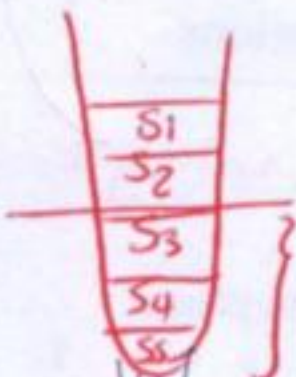
- The pyramidal tract (corticospinal tract).
- The extrapyramidal tracts.
- The cerebello-spinal tracts.

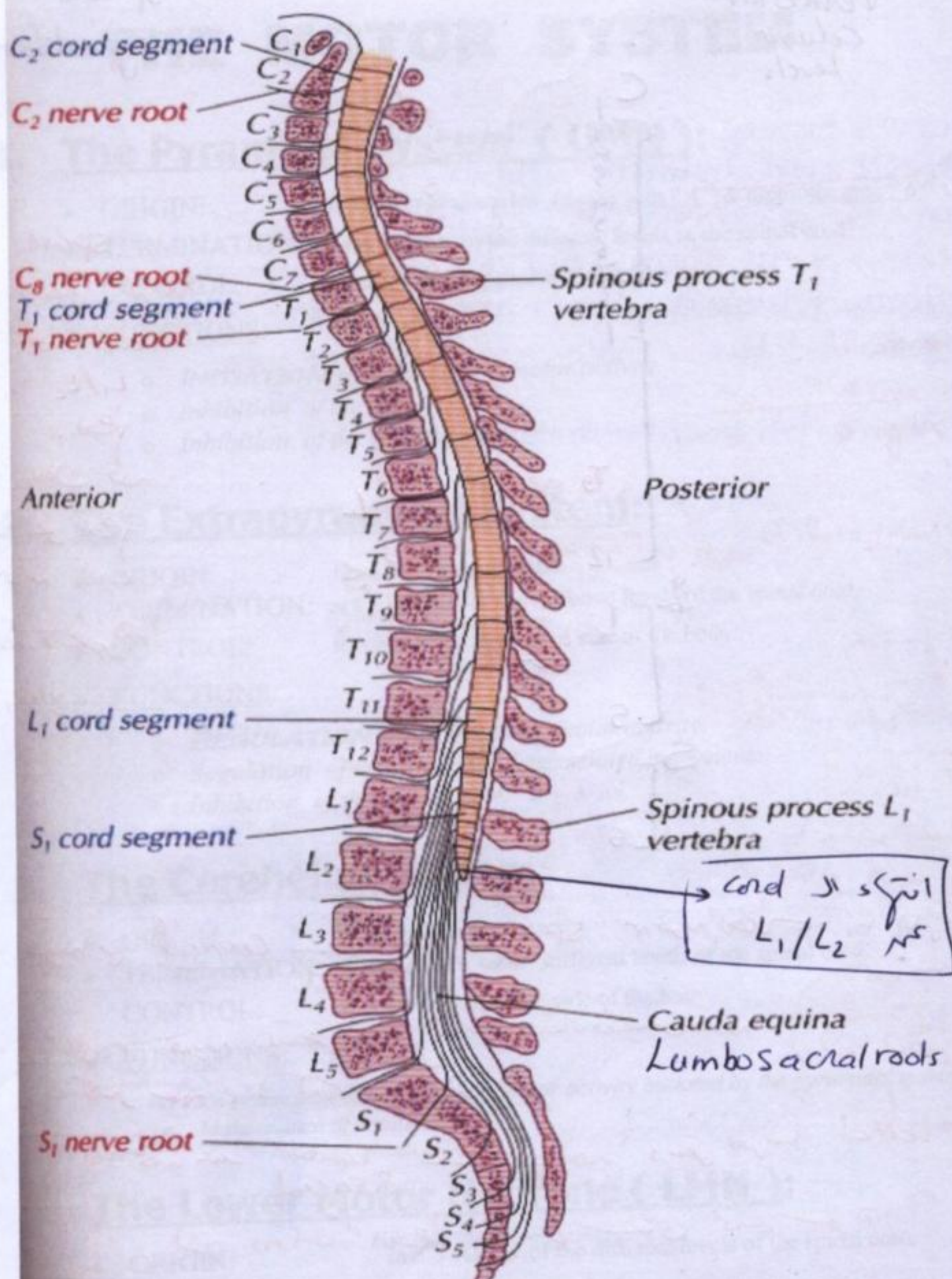
نازل من
يعلمك العضلات (الاتزان)
Cerebellum

2. Cauda Equina:

“Collection of Lumbo – sacral roots”

- o It fills the lower part of the spinal canal.
- o It starts at the lower border of the 1st lumbar vertebra (L1).

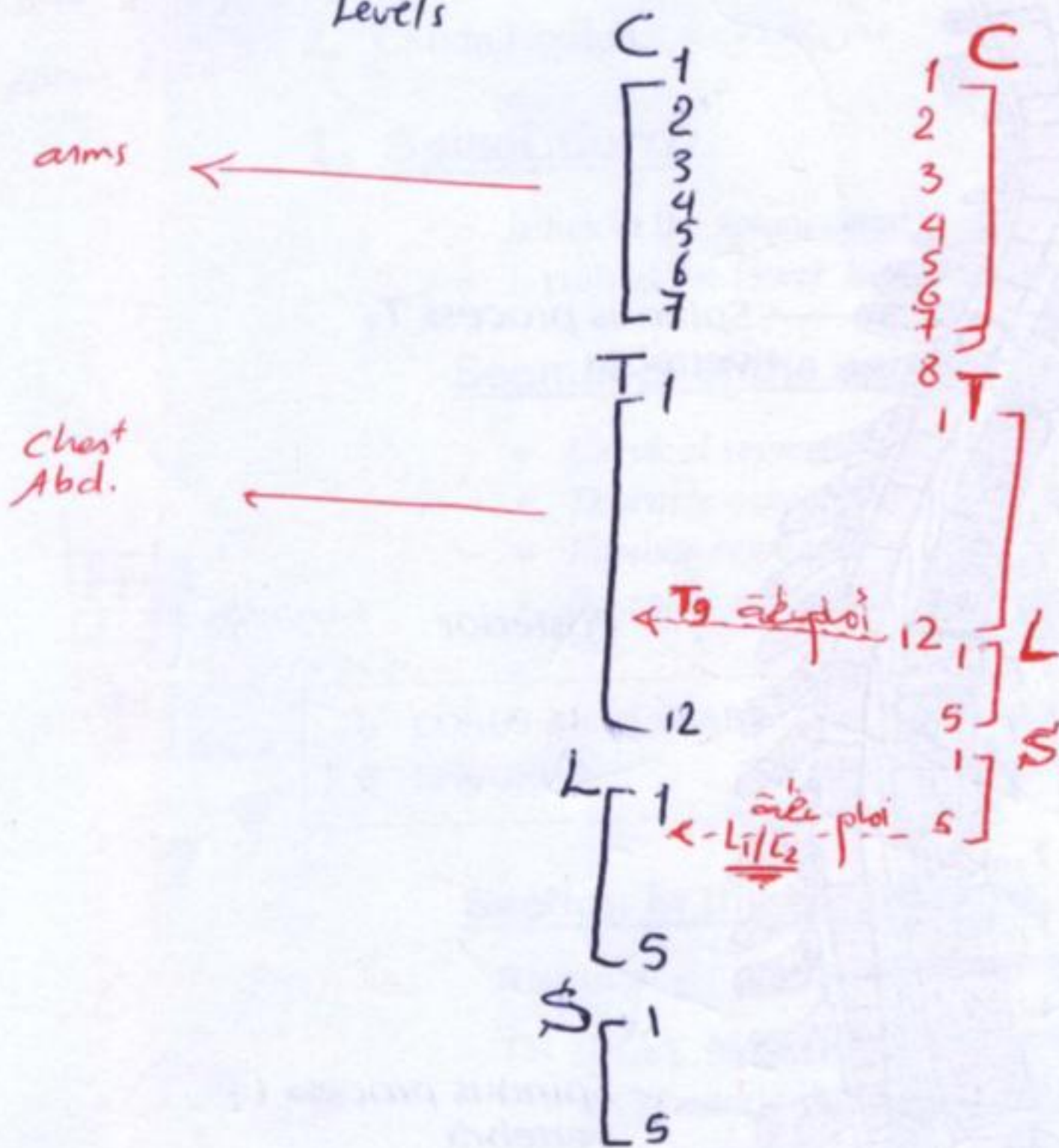




THE SPINAL CORD, LATERAL VIEW

عقود
Vertebral
column
levels

عقود شوكية
sp cord
segment levels



sp cord
واحد في الارتفاع
الى level
L1/L2
في العظم
وذلك لانه
سرعة نمو العظم
اكثر من cad
ولذلك لا يغير هذا
في البداية

* فقرة في Cervical ← يطرح sp nerve root من فقرة لاحقة اما الباقية تحت إبطية بسبب الزاوية

* roots فقرة يتبقى horizontal عتاء ففيس فارم كبير بين L1-L5 والعظم

اما تحت عند lumbar & sacral ← يحل حدة ويبقى تقريبا vertical

لانه تحتات العظم يتابعه في مستوى تحت شوك

منجلوا Cauda equina

THE MOTOR SYSTEM

1. The Pyramidal System (UMN):

- **ORIGIN:** in the cerebral cortex (motor area "4" & premotor area "6").
- **TERMINATION:** at the AHCs of the different levels of the spinal cord.
- **CONTROL:** it controls the opposite side of the body.
- **FUNCTIONS:**

الزعم
احداث التوتر

- **INITIATION** of the voluntary motor activity.
- **Inhibition** of the deep reflexes.
- **Inhibition** of the muscle tone.

weakness
tone
reflex

2. The Extrapyramidal System:

- **ORIGIN:** from the basal ganglia.
- **TERMINATION:** at the AHCs of the different levels of the spinal cord.
- **CONTROL:** it controls the opposite side of the body.
- **FUNCTIONS:**

Caudate
Putamen
Globus pallidus

+ help of thalamus

تنظيم التنفيس

- **REGULATION** of the voluntary motor activity.
- **Regulation** of the emotional & associated movements.
- **Inhibition** of the muscle tone.

Breaking
مفترقة

but no
inhibitory
reflexes

invol. movement
حيث لو مش
عائز تحرك يدك
هو يحافظ على عتاه
متحركش لو باطله

3. The Cerebellar System:

- **ORIGIN:** from the cerebellum.
- **TERMINATION:** at the AHCs of the different levels of the spinal cord.
- **CONTROL:** it controls the same side of the body.
- **FUNCTIONS:**

donot
cross

- **Co-ordination** of the voluntary motor activity initiated by the pyramidal system.
- **Maintenance** of equilibrium.

مثل حركة
اليدين أثناء المشي
لنلا لو باطل
loss of swinging
of arms
during walk

4. The Lower Motor Neurone (LMN):

- **ORIGIN:** in the AHCs of the different levels of the spinal cord.
- **TERMINATION:** at the voluntary muscles.
- **COMPONENTS:** AHCs, PN, NMJ & Voluntary muscles.
- **FUNCTIONS:**

- **Transmission** of the motor impulse from the AHCs to the voluntary muscles.

حركة
ناعجة
هو جبهة
الى الامكان
الرفع

باطله
نطلع الحركة
incoordinated

MND

G.B.S

Myasthenia

Myopathy

UMNL
opp- side

LMNL
Same side

Cr 7

Pons

الطرف
اليسرى
واحده
منه

Cr 12

Medulla

if lesion in area 4 (UMNL)

R+ 7
المنطقة

عبرته من المنطقة

R+ 12

THE MOTOR SYSTEM

كل البوابة دي عناء حركة العضلة
 . وبنه لو الإشارة من Δ الى العضلة

صيعل حركة
 من مضبوط

للأزم يعنى
 على

E.A
 . Cerebell.

The Pyramidal System
 "Controls **opposite** side"

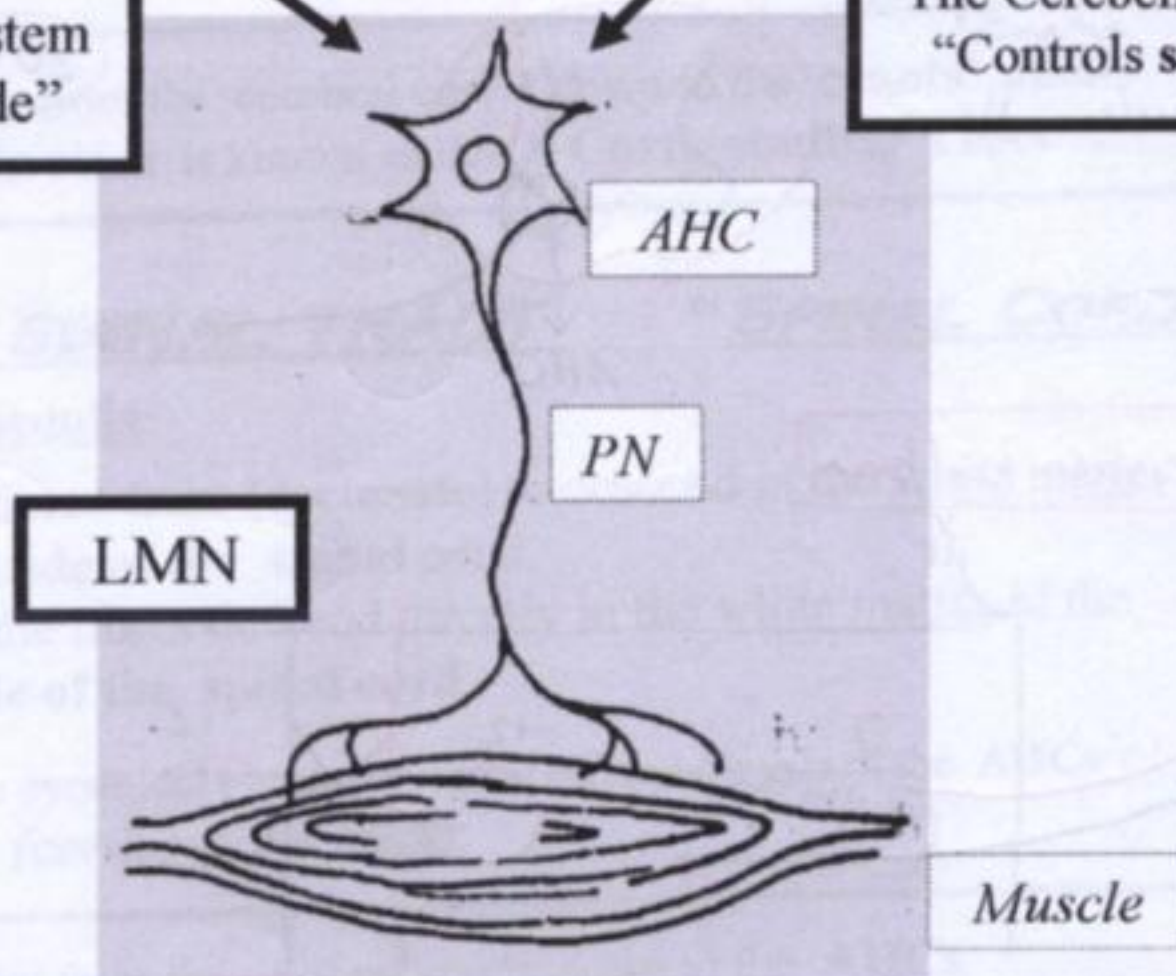
Crossing at
 lower medulla
 to AHCs

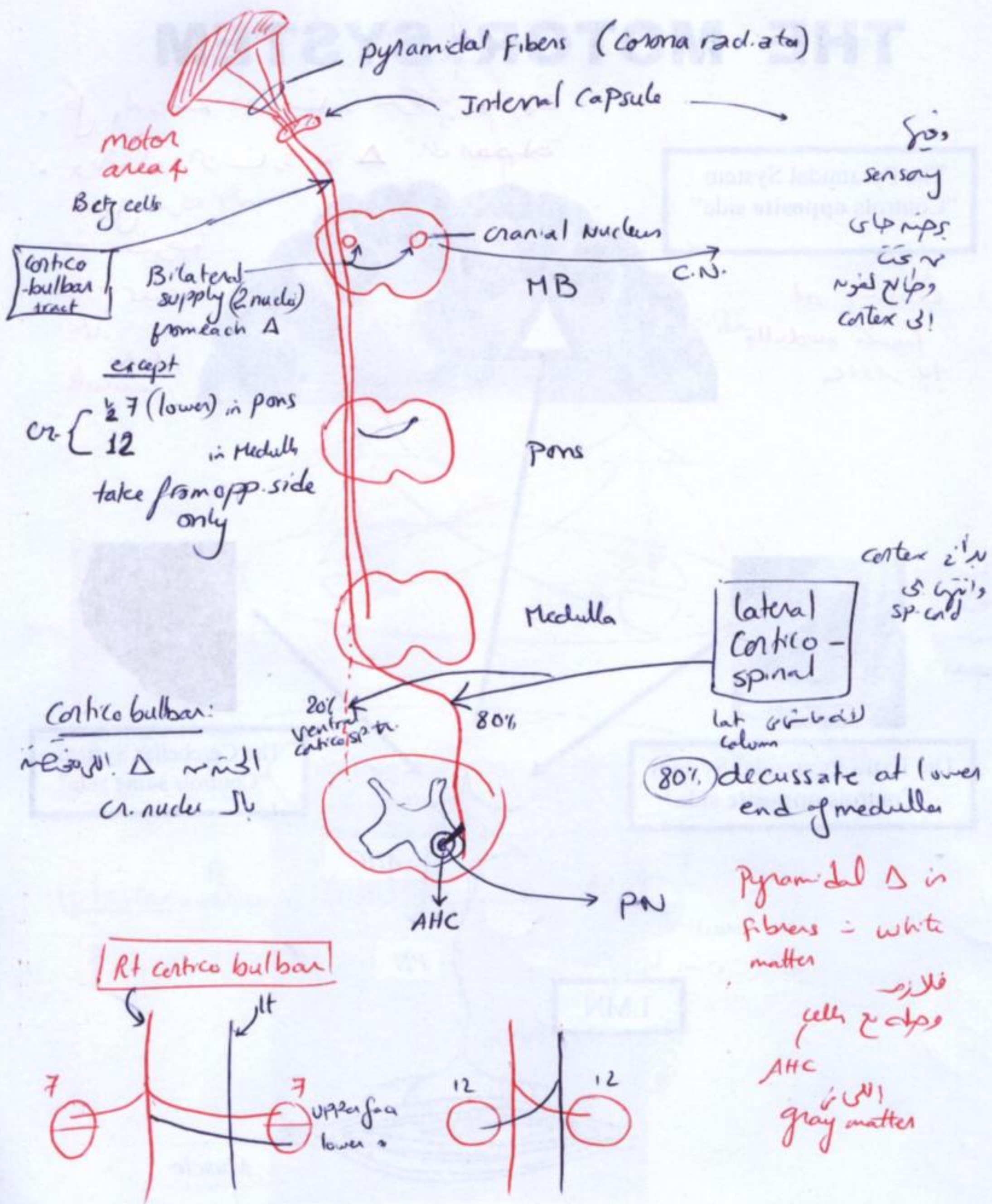


The Extra Pyramidal System
 "Controls **opposite** side"



The Cerebellar System
 "Controls **same** side"





لو عازر ابرخه نفس الوش
الرفوف لازم اضر الناحیه
لو عازر ابرخه نفس الوش
الرافت کفایه اضر الناحیه
opp. side

MOTOR PATHWAY

- ORIGIN: the order starts in the Cerebral cortex. *area 4*
- TERMINATION: on the Voluntary muscle which will respond by movement.

From the origin to the termination, the impulse passes through 2 neurones:

1. Upper Motor Neurone (UMN): PYRAMIDAL TRACT.
2. Lower Motor Neurone (LMN): AHCS, Nerves, NMJ, Muscles.

1. UPPER MOTOR NEURONE (UMN)

1. MOTOR AREA "4" "CEREBRAL CORTEX"

The voluntary motor impulse originates mainly in the large pyramidal cells (Betz cells) of the motor area "4" & to a lesser extent in the cells of the premotor area "6".

2. INTERNAL CAPSULE

The **axons** of these cells (carrying the impulse) descend in the depth of the Cerebral hemisphere in the Corona radiata to pass in the Internal capsule, and, continue their descent in the brain stem (midbrain, pons & medulla).

3. CORTICOBULBAR TRACT "BRAIN STEM"

In the Brain Stem, some of the descending fibres separate to supply the motor nuclei of the cranial nerves of BOTH sides except the lower 1/2 of the facial nucleus and all the hypoglossal nucleus which are supplied only from the opposite pyramidal tract.

These fibres are known as the corticobulbar tract as they do not reach the spinal cord.

The pathway from the cerebral cortex down to the **cranial nuclei** in the **brain stem** is known as the "**Corticobulbar Tract**".

4. CORTICO SPINAL TRACT "SPINAL CORD"

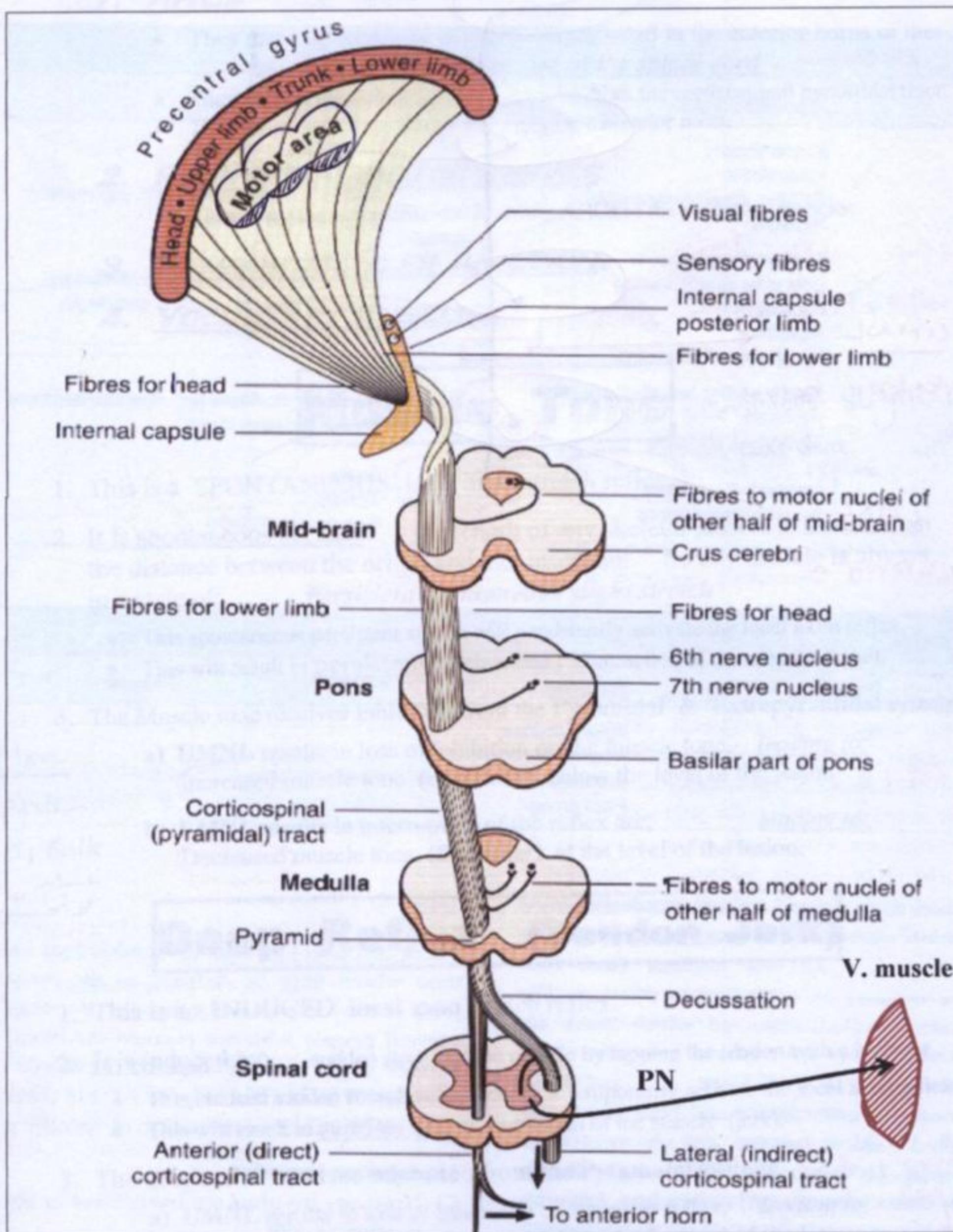
In the lower medulla:

- 80 % of fibres cross (decussate) to descend in the white matter of the **opposite side of the spinal cord**,
- 20 % of the fibres descend directly in the white matter of the **same side of the spinal cord**.

The fibres of the pyramidal tract terminate at different levels of the AHCs of the spinal cord (corticospinal tract).

The pathway from the cerebral cortex down to the **AHCs** in the **spinal cord** is known as the "**Corticospinal tract**".

Pathway of the voluntary motor impulse (UMN & LMN)



2. LOWER MOTOR NEURONE (LMN)

1. AHCs

- They are: special type of nerve cells situated in the anterior horns of the: "H-shaped gray matter of the spinal cord".
- They receive the voluntary motor impulse from the corticospinal pyramidal tract.
- Their axons exit from the spinal cord as the anterior roots.

2. PERIPHERAL MOTOR NERVES

- They carry the motor impulse from the AHCs to the voluntary muscles.

3. NEUROMUSCULAR JUNCTION.

4. VOLUNTARY MUSCLES.

Oral reflex شىء مؤتمت
Tone reflex دائماً مستمر هو Tone

Muscle Tone

= continuous slight contraction
موتد مستمر
persistent reflex

1. This is a SPONTANEOUS local axon stretch reflex.
2. It is spontaneous because: the length of any skeletal muscle is shorter than the distance between the origin and the insertion. So any muscle is always in a state of: **Persistent spontaneous slight stretch**.
 - This spontaneous persistent stretch will persistently activate the local axon reflex.
 - This will result in **persistent** (maintained) contraction of the muscle (tone).
3. The Muscle tone receives inhibition from the Pyramidal & Extrapyramidal systems:

Fin of tone

- 1- Posture
 - 2- Its Bulk
- عنه
لا تحس

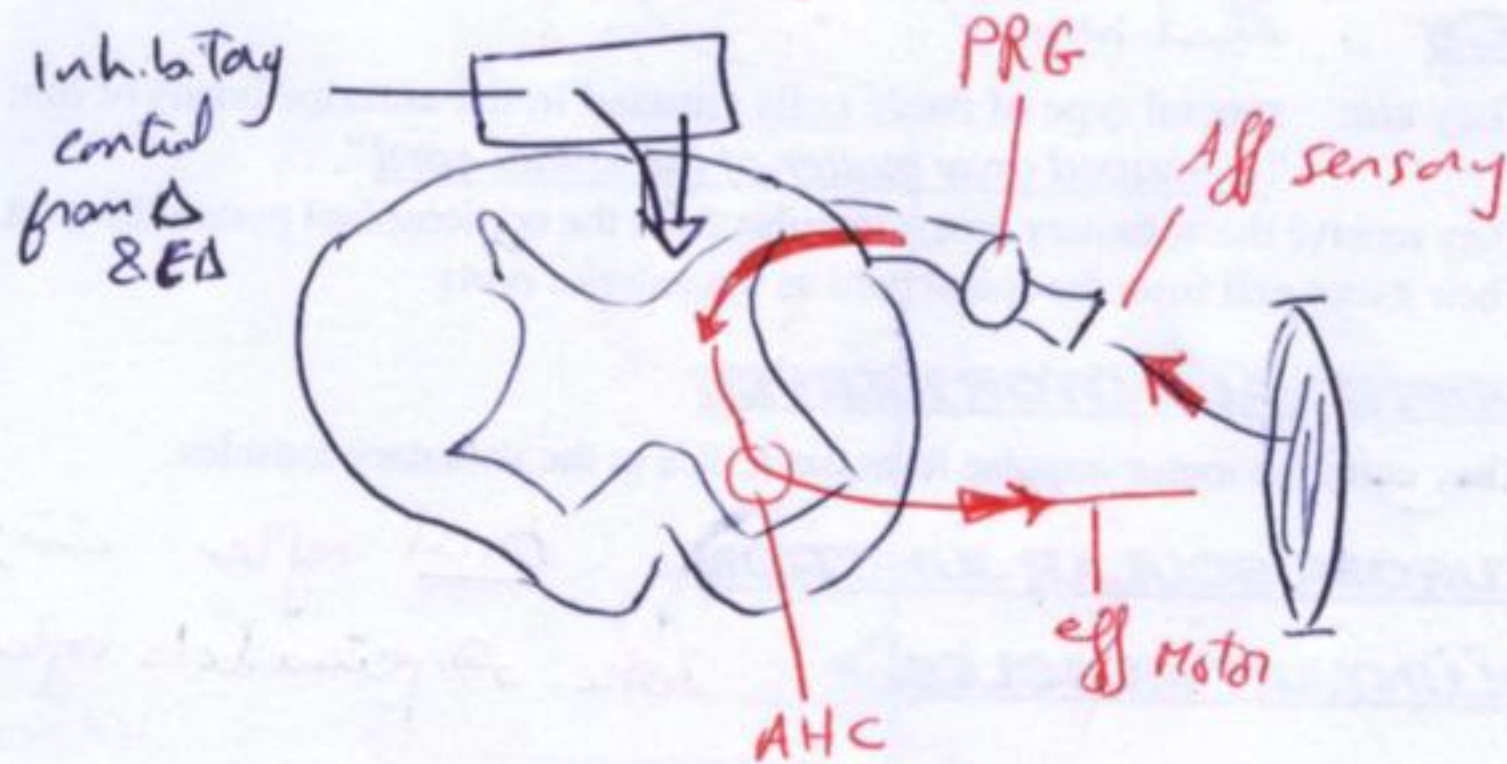
- a) **UMNL** results in loss of inhibition on the muscle tone, leading to:
Increased muscle tone (spasticity) below the level of the lesion. *if EA rigidity*
- b) **LMNL** results in interruption of the reflex arc, leading to:
Decreased muscle tone (flaccidity) at the level of the lesion.

Deep Reflex (Tendon Jerk)

1. This is an INDUCED local axon stretch reflex.
2. It is induced by: sudden stretch of the muscle by tapping the tendon with a hammer.
 - This induced sudden stretch will suddenly & temporarily activate the local axon reflex.
 - This will result in **sudden sharp** contraction of the muscle (jerk).
3. The deep reflex receives inhibition from the Pyramidal system:
 - a) **UMNL** results in loss of inhibition on the deep reflex, leading to:
Increased deep reflex (**hyperreflexia**) below the level of the lesion.
 - b) **LMNL** results in interruption of the reflex arc, leading to:
Decreased deep reflex (**hyporeflexia**) at the level of the lesion.

if severe lesion
↓
Clonus Pathological
anterior pituitary

ای عضلہ کے جسم طویل اقصیٰ سے ملتا ہے۔
 0 & I
 لڑاکے ہیں
 عضلہ کے طول سے ← یہی reflex کے طول



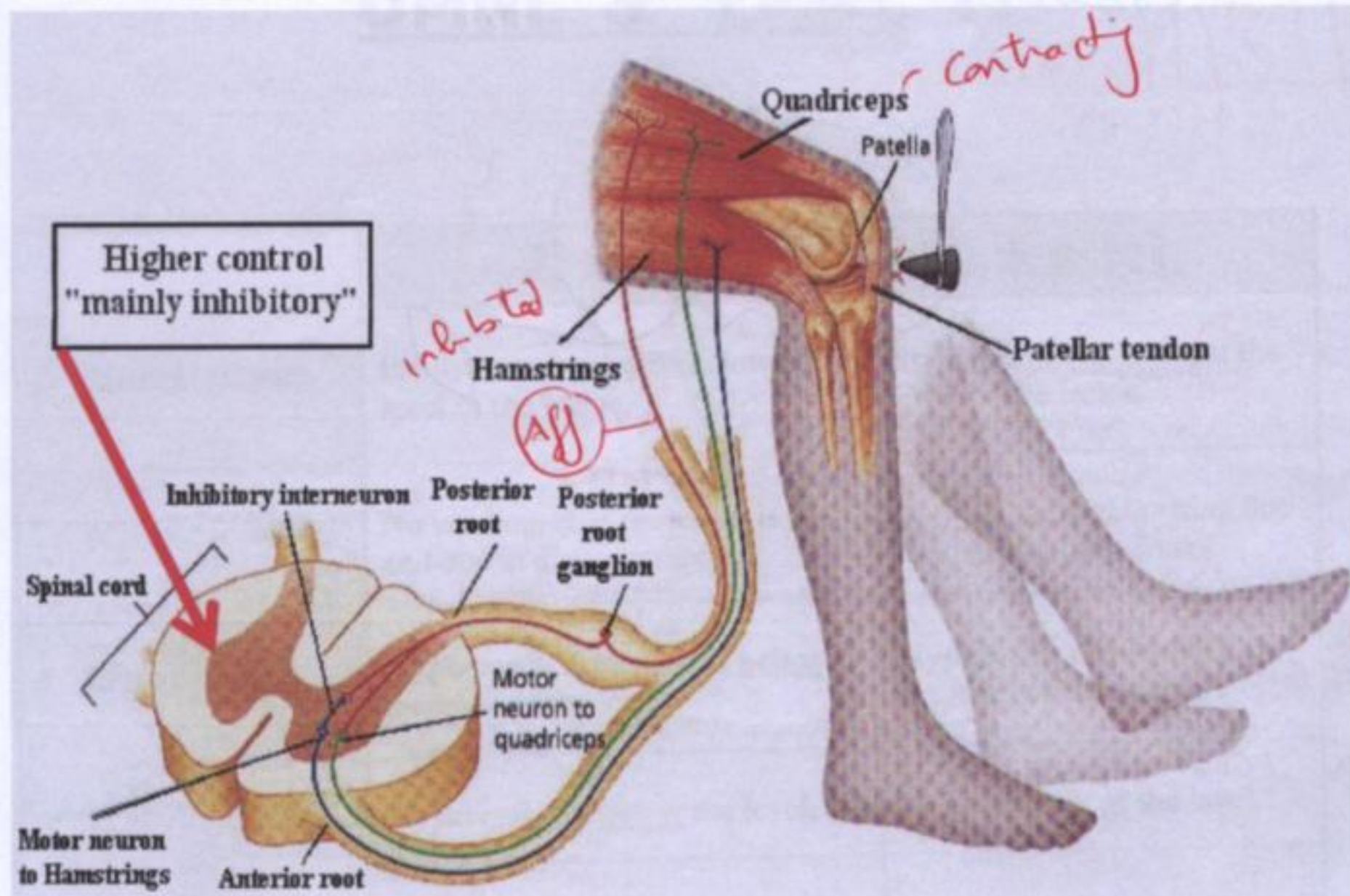
Tone deep

Scale
 micturition
 Local axon
 brain
 by stretch

Deep Reflex (Tendon Jerk)

STRETCH REFLEX

(Muscle tone & Deep reflex)



Clonus

- Sudden sustained stretch of the muscle tendon, results in:
Rapid, Rhythmic, Regular contractions.

1. It indicates: Severe pyramidal lesion due to: loss of inhibition on the stretch reflex.
2. It is elicited in the: ankle, patella, wrist.
3. It stops when: the stretch is stopped.

Clonus & Babinski

خلفه
مفرد

10

How would you clinically differentiate between:

UMNL & LMNL ??

زی امهك بالحیظ

severity

	UMNL	LMNL
1. Muscle power	Paralysis or weakness <u>below</u> the level of the lesion.	Paralysis or weakness <u>at</u> the level of the lesion.
2. Muscle wasting	No wasting, & if present it is late and due to disuse atrophy. <i>hypertonia</i>	<u>Early & marked</u> wasting due to loss of muscle <u>tone</u> .
3. Muscle tone	<u>Hypertonia</u> (spasticity) <u>below</u> the level of the lesion.	<u>Hypotonia</u> (flaccidity) <u>at</u> the level of the lesion.
4. Deep reflexes	<u>Hyperreflexia</u> <u>below</u> the level.	<u>Hyporeflexia</u> <u>at</u> the level.
5. Pathological deep reflexes	May be present.	Absent.
6. Clonus	May be present. ✓	Absent.
7. Superficial reflexes	Lost if the lesion is above the segmental supply of the reflex.	Lost if the lesion involves the supply of the reflex.
8. Plantar reflex (Babinski)	<u>Positive</u> , i.e. dorsiflexion of the big toe ± fanning of the other toes. <i>normal: plantar flexion</i>	Plantar flexion of the toes, or <u>no response</u> . <i>فردی</i>
9. Fasciculations	Absent	May be present in irritative lesion of the <u>AHCs</u> .



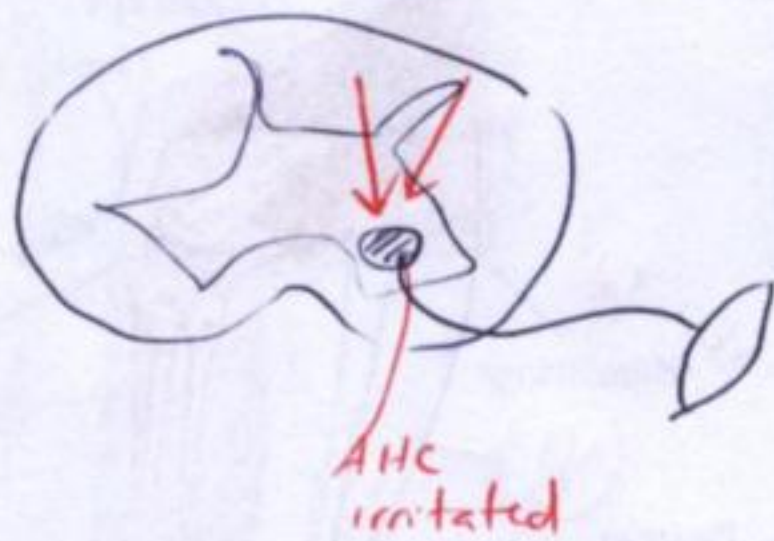
specific to Δ

but not sensitive as also in children, coma

10

Fasciculation = LMNL (AHC)

العضلة تفسر ترقص من ألياف
عنه تتحرك انفرادي
العضلة



Clonus

1. It indicates
2. It is related to the
3. It stops when the stretch is stopped
4. It indicates
5. It is related to the
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100. It indicates

THE SENSORY SYSTEM

- There are 3 types of sensations in the body:

I. SOMATIC SENSATIONS:

- * They are conducted to the CNS via the "somatic nerves."
- * They include:

1. Superficial sensations: *pin prick*

- Pain.
- Temperature.
- Touch. *fine crude*

يقول أي لورميك
thalamus

2. Deep sensations (proprioceptive):

- Vibration sense. *tuning fork*
- Joint sense. *position*
- Muscle sense.
- Nerve sense.

يس لورميك
thalamus

أكثر واحد
reliable
vibration sense

3. Cortical sensations:

- Tactile localization.
- Two points discrimination.
- Stereognosis.
- Graphosthesia.

يس لما توصل
thalamus

لما تقاويل الركة
في ادايه ومنه
عبره
area
1, 2, 3

اديله حايه
معرفة يرك
اكتب ماديده رقم كير هير

II. VISCERAL SENSATIONS:

- * They are conducted to the CNS via the "autonomic nerves."
- * They include all sensations coming from the internal viscera,
e.g. *heart & intestines.*

سوي
اعلى وأرقى

III. SPECIAL SENSATIONS:

- * They are conducted to the CNS via the "cranial nerves."
- * They include: *Smell, Vision, Hearing.*

Somatic Sensations

- All somatic sensations, whether superficial or deep pass through **3 ORDER NEURONES** from receptors in the skin & deep structures to reach the cortical sensory area of the opposite side.

1. The cell of 1st order neurone is: always in the posterior root ganglion of the same side.
2. The cell of 3rd order neurone is: always in the thalamus of the opposite side.
3. The cell of 2nd order neurone: varies according to the type of sensation.

A) PATHWAY OF SUPERFICIAL SENSATIONS

سؤال
anatomy
سؤال

I. Pathway of Pain & Temperature:

1. The 1st order neurone:

- The 1st order neurone is the cell of the **posterior root ganglion (PRG)**.
- The afferent sensory nerve relays in the PRG.
- The efferent sensory nerve enters the spinal cord in Lissauer's tract to relay in the cells of the **Substantia Gelatinosa of Rolandi (SGR)**, at the posterior horn of the gray matter.

2. The 2nd order neurone:

- The 2nd order neurone is the cell of **SGR** & its axon.
- This axon crosses to the **OPPOSITE** side & ascends in the: **Lateral Spinothalamic tract** of the spinal cord, then in the: **Lateral Lemniscus** of the brain stem, to relay in the thalamus.

3. The 3rd order neurone:

- The 3rd order neurone starts in the cell of the **thalamus**.
- Its axon ascends to pass through the internal capsule conducting the impulse to the cortical sensory area in the parietal lobe.

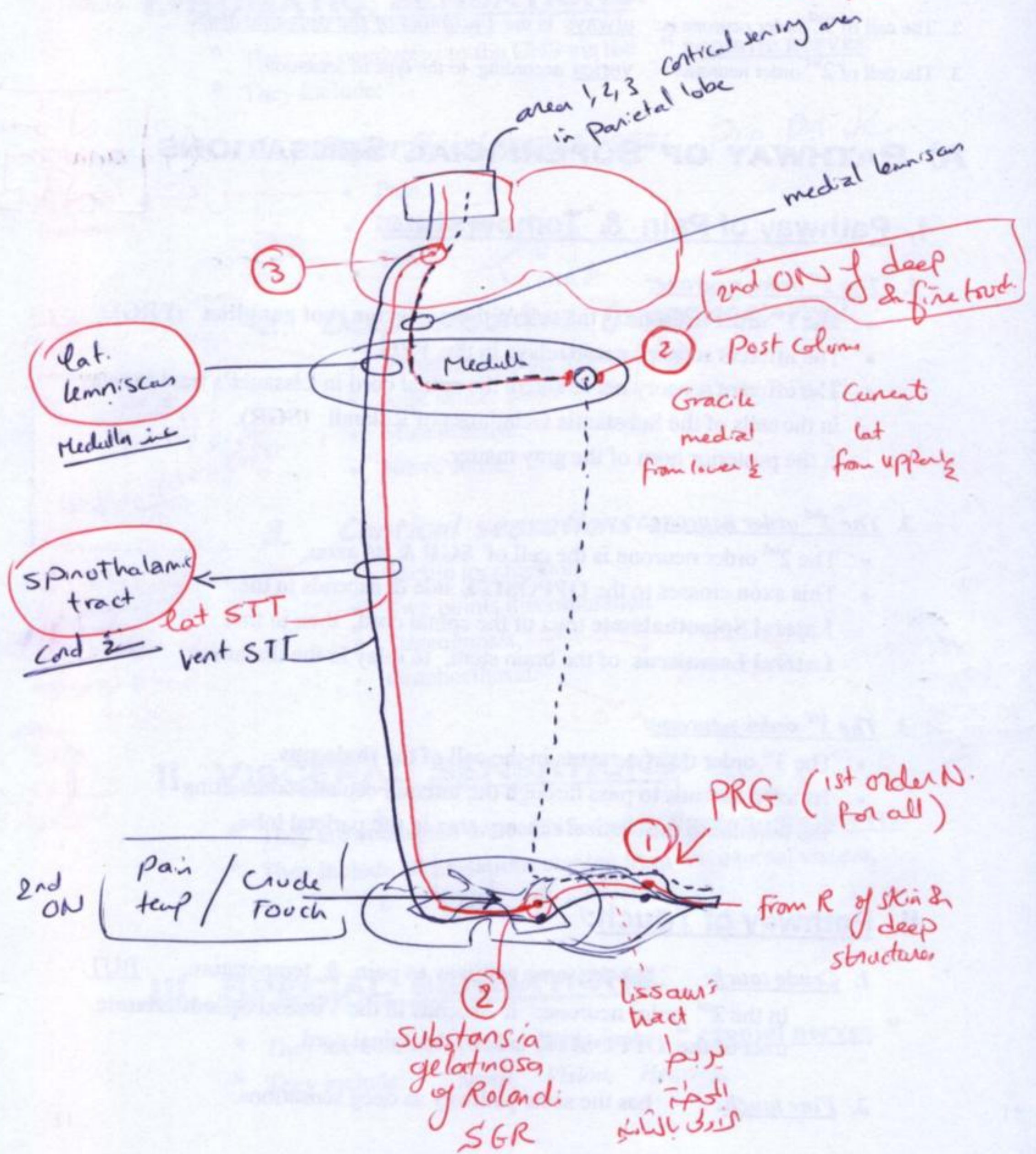
II. Pathway of Touch:

1. Crude touch: has the same pathway as pain & temperature, **BUT:**
In the 2nd order neurone: it ascends in the **Ventral Spinothalamic tract** of the **OPPOSITE** side of the spinal cord.

2. Fine touch: has the same pathway as deep sensations.

(relay) محطات ٢ من sensory pathway superficial deep

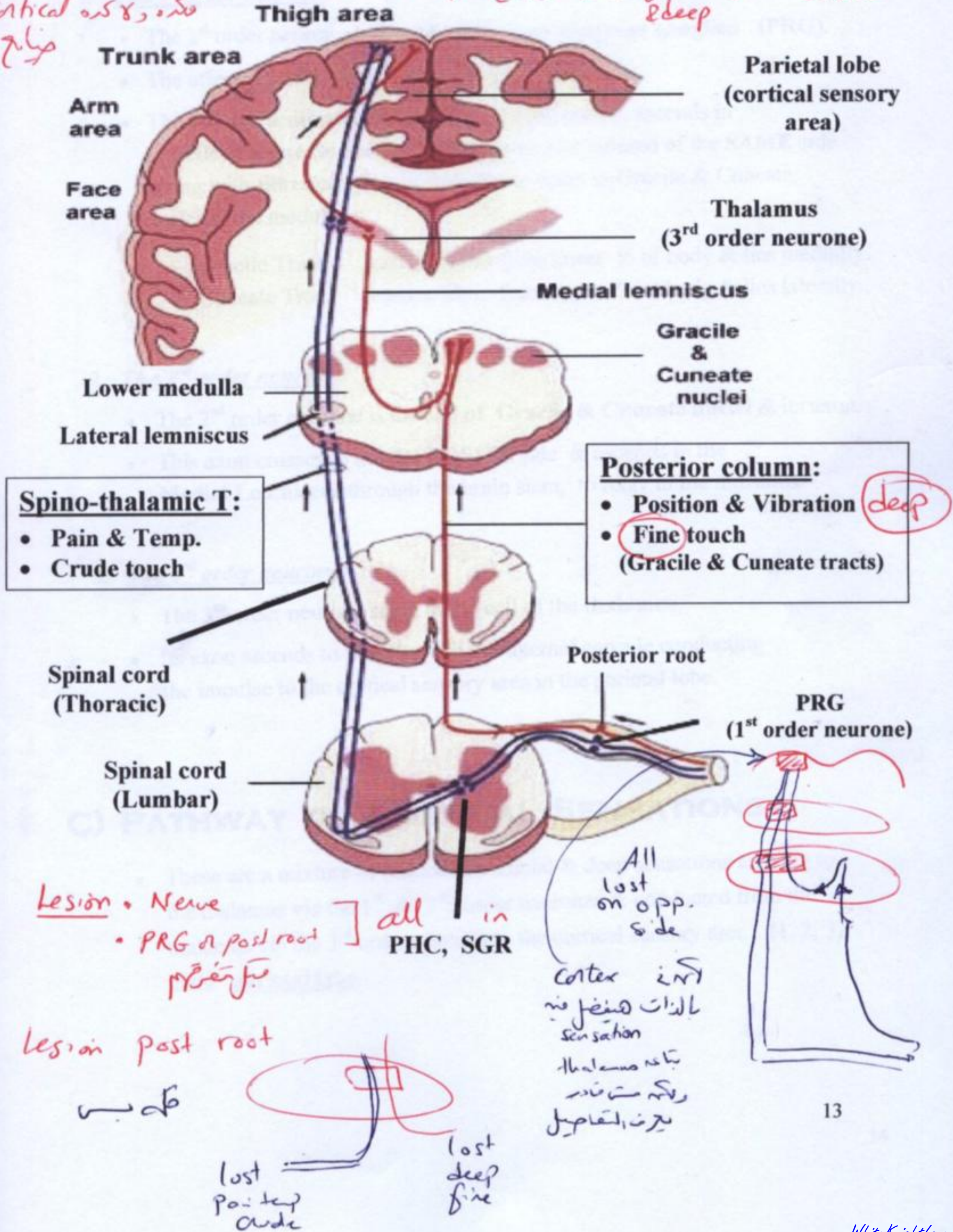
كلهم يتفقون على المحطة الأولى والثالثة ويختلفون في الثانية.
 المحطة الأولى: مركزية بحتة. المحطة الثانية: thalamus وليس بحتة. المحطة الثالثة: thalamus وليس بحتة.



Pathway of Superficial & Deep Sensations

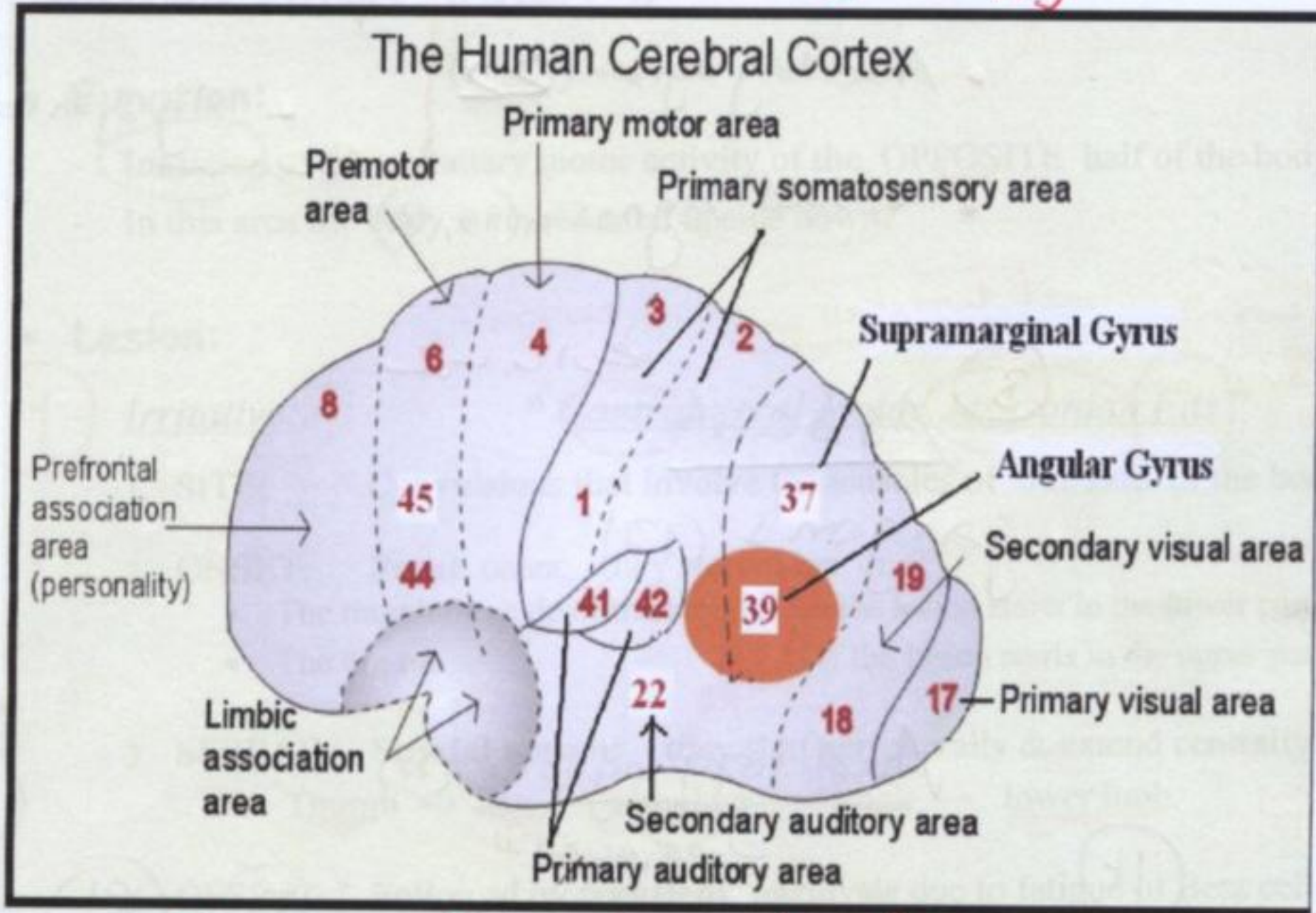
Thalamus level lesion
Cortical area
Fine & deep
Cortical sensory area

oral
لا يسمع اختبار
ال
Cortical sensory



Areas of the Cerebral Cortex

for $\left\{ \begin{array}{l} \text{tumors} \\ \text{blood supply diseases} \end{array} \right.$



* if area : (only in dominant hemisphere)
عكس الناحية التي يمكنه
(if hemisphere)

لويحات
نقل
aphasia

لو من مكتوب
أمره
بilateral

The surface of each cerebral hemisphere is divided into 4 LOBES:

1. **Frontal.**
2. **Parietal.**
3. **Temporal.**
4. **Occipital.**

Each lobe consists of: MANY AREAS.

Each area is responsible for a specific function.

* Lesion $\left\{ \begin{array}{l} \text{irritative (early)} = \text{stimulatory} \\ \text{destructive (late)} \end{array} \right.$

++fn $\rightarrow$ لوظائف
أدى
--fn $\rightarrow$ تلف

FRONTAL LOBE

1. MOTOR AREA (AREA 4)

▪ Function:

- Initiation of the voluntary motor activity of the **OPPOSITE** half of the body.
- In this area the body is represented **upside down**.

▪ Lesion:

1- Irritative:

"Contralateral Motor Jacksonian Fits"

1. SITE: Convulsions that involve the muscles of **one side** of the body.
2. ONSET: **Focal onset**, they start either in:
 - The thumb or **angle** of the mouth (if the lesion starts in the lower part).
 - The big toe (if the lesion starts in the upper part).
3. SPREAD: **Special march**: they start peripherally & extend centrally:
 - **Thumb** → **arm** → **shoulder** → **trunk** → **lower limb**.
4. OFFSET: Followed by **transient paralysis** due to fatigue of Betz cells:

- Todd's paralysis.

not more than 24 hrs

2- Destructive:

= Betz cell
حصول فيزي تلف

"Contralateral Motor Paralysis"

Permanent Irreversible Paralysis

Oral

حيز أسباب إكل مؤقت
Causes of
transient
epilepsy

بطل الاعيان طول
عمر عاجز الا لوط

(paralysis →
(مشلل))

دواء
Physiotherapy

2. PREMOTOR AREA (AREA 6)

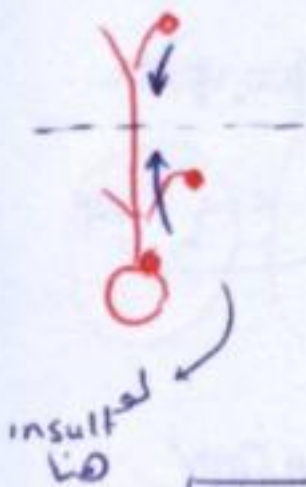
▪ Function:

Inhibition of tone & reflex

- Partly supplies the pyramidal tract.
- Partly supplies the extra-pyramidal tract.

▪ Lesion:

- Contralateral Hypertonia & Hyperreflexia.



علاج
نوع رجيم
تختلف القولبي
destruction

3 ← مائة العلاقات
4 ← بحجة رقت 50
6 ← ليرة LR
أما إلى يترك الحية لوحدها هو

17

3. AREA OF CONJUGATE EYE DEVIATION (AREA 8)

Function:

- Voluntary conjugate eye deviation to the OPPOSITE side.

Lesion:

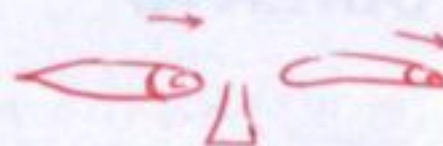
Irritative:

- Attacks of conjugate eye deviation to the OPPOSITE side of the lesion.

Destructive:

- Loss of conjugate eye deviation to the OPPOSITE side of the lesion.

Other area moves
2 eyes
conjugately
by (HLB)
also in pons



4. BROCA'S AREA (44)

"In The Dominant Hemisphere Only"

Function:

- It is the Motor center for speech.
- It is responsible for formulation of spoken words.
- It is present in the left cerebral cortex in right-handed persons & vice versa.

Lesion: "Motor Aphasia (Verbal Aphasia)"

- The patient cannot express himself in spoken words.

أحوال أفكار مضمي إلى كلام
إصدار الفكرة

أما حركة
اللسان والفم
↓
by area 4
(Cortico bulbous)



5. EXNER'S AREA (45)

"In The Dominant Hemisphere Only"

Function:

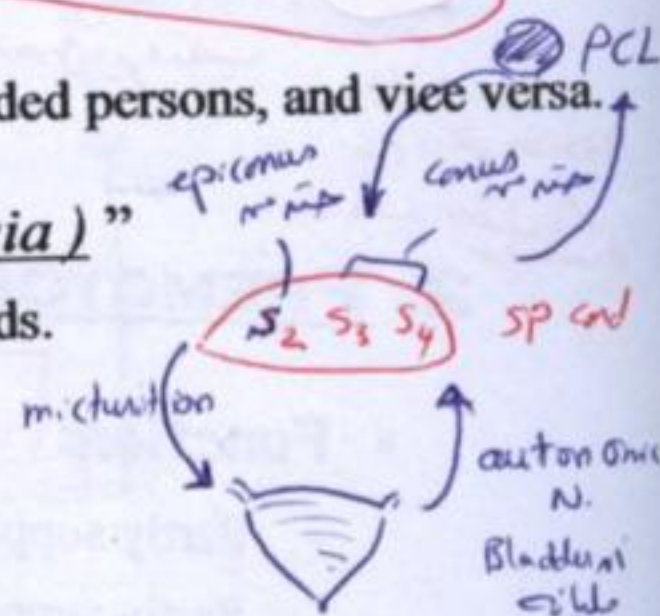
- It is the motor center for writing.
- It is responsible for formulation of written words.
- It is present in the left cerebral cortex in right-handed persons, and vice versa.

Lesion: "Agraphia (Writing Aphasia)"

- The patient cannot express himself in written words.

لو اسلك اي؟ ← صير مشي لانه الفكرة لم تتوحي (ولكنه يقدر يكتب) ورنه (aphasia)
لو اسلك اي؟ ← اي ... ولكنك مجتهد وحسن (dysgraphia)

لم أتوحي الفكرة
لكنك ...
أما لو لم أتوحي
أفعل ...
ردده
area 4
أخرى
↓
Sensory aphasia



6. PARACENTRAL LOBULE

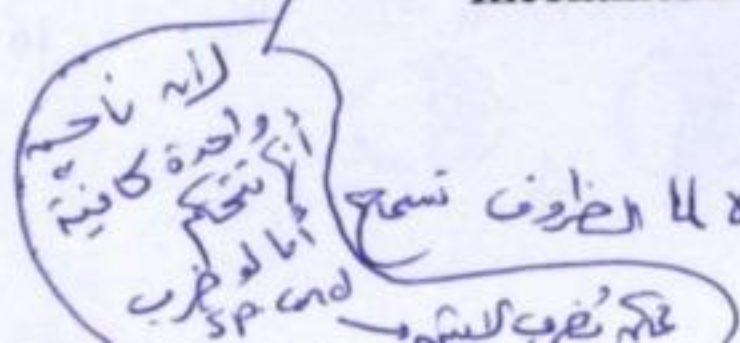
Function:

- Cortical inhibition (control) of bladder voiding & bowel evacuation.

Lesion: (if Bilaterally)

- Incontinence of urine & stools.

← عن طريق SP cord لا يتبول تلقائياً
الآن أطفال وحيوانات



higher centre
adult human
inhibition ويقل SP cord

17

7. PREFRONTAL AREA

Function:

- It is the higher center for:
 - o Mentality (Memory & Intelligence).
 - o Personality (Behaviour, Planning, Problem - solving).
- It inhibits the primitive reflexes present in the newborn, e.g. grasp reflex.

Lesion:

- Disturbances in: Mentality & Personality → DEMENTIA.
- Reappearance of the primitive reflexes, e.g. grasp reflex.

PARIETAL LOBE

1. CORTICAL SENSORY AREA (1, 2, 3)

Function:

- Perception of cortical sensations from the OPPOSITE half of the body.
- In this area the body is represented upside down.

Lesion:

Irritative:

1. SITE: Sensory fits (numbness or tingling) in one side of the body.
2. ONSET: Focal onset.
3. SPREAD: Special march.

Destructive:

"Contralateral Cortical Sensory Loss"

2. ANGULAR GYRUS (39)

"In The Dominant Hemisphere Only"

Function:

- It is responsible for Reading, i.e. Recognition & Recall of letters & numbers.

Lesion:

"Alexia (Visual Aphasia, Word Blindness)"

- The patient is able to see the letters & numbers, BUT, THEREFORE, He is not able to recognize (understand) them, He is not able to read.

Alexia
كل ما يكتبه
بالجريبكي
كأنه الكلام أي لغة ثانية أنا من فاهي

ولد = boy
خزنت
الاسم والمعنى مرتبطاً به



3. SUPRAMARGINAL GYRUS (37) "In The Dominant Hemisphere Only"

▪ Function:

- It is responsible for:

Storage & Recall of:

1. IDEAS of speech.

2. IDEAS of complex voluntary motor activity.

▪ Lesion:

1. Jargon's Aphasia (Word salad).

2. Apraxia: inability to perform complex voluntary motor activity, in absence of: paralysis, sensory loss or incoordination.

TEMPORAL LOBE

1. PRIMARY AUDITORY AREA (41, 42)

▪ Function:

- It is the center for HEARING.

▪ Lesion:

- Irritative: هلوسة أصوات مستمرة موجودة
o Auditory Hallucinations.

- Destructive:

o Slight impairment of HEARING.

o Never complete deafness because hearing is bilaterally represented.

= auditory association area

2. SECONDARY AUDITORY AREA (22) "In The Dominant Hemisphere Only"

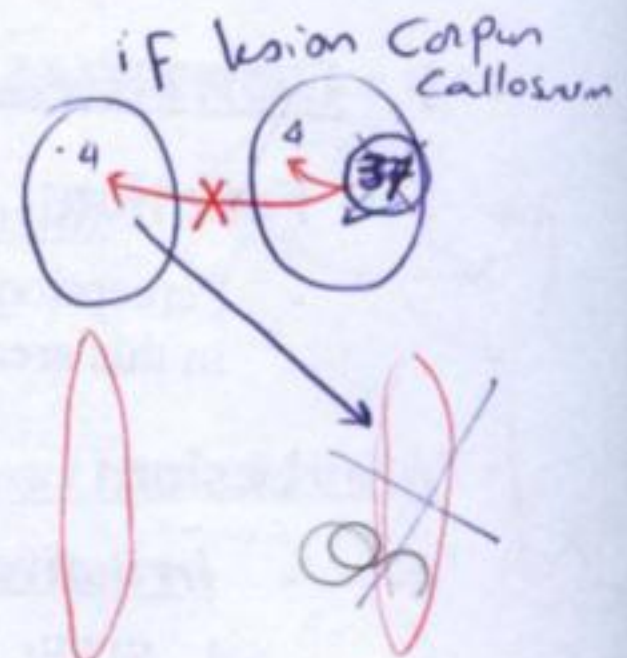
▪ Function:

- Recognition & Recall of sounds & heard words.

▪ Lesion:

"Auditory Agnosia"

- The patient is able to hear the sounds, BUT, He is not able to recognize (understand) them.



3. LIMBIC SYSTEM (UNCUS)

▪ Function:

- It is the center for SMELL.

▪ Lesion:

- Irritative:

- Olfactory Hallucinations.

- Temporal lobe epilepsy

- Destructive:

- Slight impairment of SMELL.

- Never complete anosmia because smell is bilaterally represented.

- Loss of memory of recent events (Anterograde amnesia)
فاكر زمانه. مت فاكر امبارج

(uncinate fits)

OCCIPITAL LOBE

1. PRIMARY VISUAL AREA (17)

▪ Function:

- It is the center for VISION.

▪ Lesion:

- Irritative:

- Visual Hallucinations.

- Destructive:

- Contralateral Homonymous Hemianopia (bilateral half blindness).

- Never bilateral complete blindness because vision is bilaterally represented.

Retina → O. Nerve
Chiasma
tract

(17)

السمع قاع يروج
النميتون فلا يروج
السمع ابد

هنا يروج

الرمحة حقبة فاصية

Macular spot
- light reflex spot

= Visual associative area

2. SECONDARY VISUAL AREA (18, 19) "In The Dominant Hemisphere Only"

▪ Function:

- Recognition & Recall of images.

▪ Lesion:

"Visual Agnosia"

- The patient is able to see the images, BUT,
He is not able to recognize (understand) them.

ما في باسوف
مشوف جديد عكاش (لم اتعرف عليه رغم اني كت عارنه ميزه)

Aphasias

1

Sensory

مستعارف
اتلقى

• Alexia (39)
مستعارف يتلقى الكلام بالقرارة

• Auditory agnosia (22)

• Visual agnosia (19, 18)

2

Motor

مستعارف الملع

• Verbal (44)
مستعارف يتكلم

• Agaphia (45)
مستعارف يكتب

3

Associative

مستعارف اربط

• Jargon's (37)

~~Auditory agnosia (22)~~

~~لا ايترون في ملاحظات~~

11

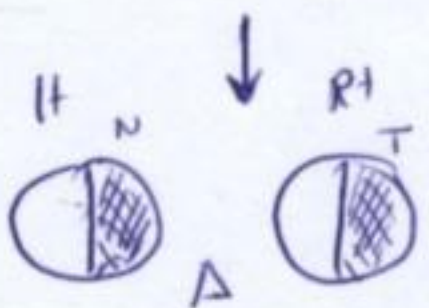
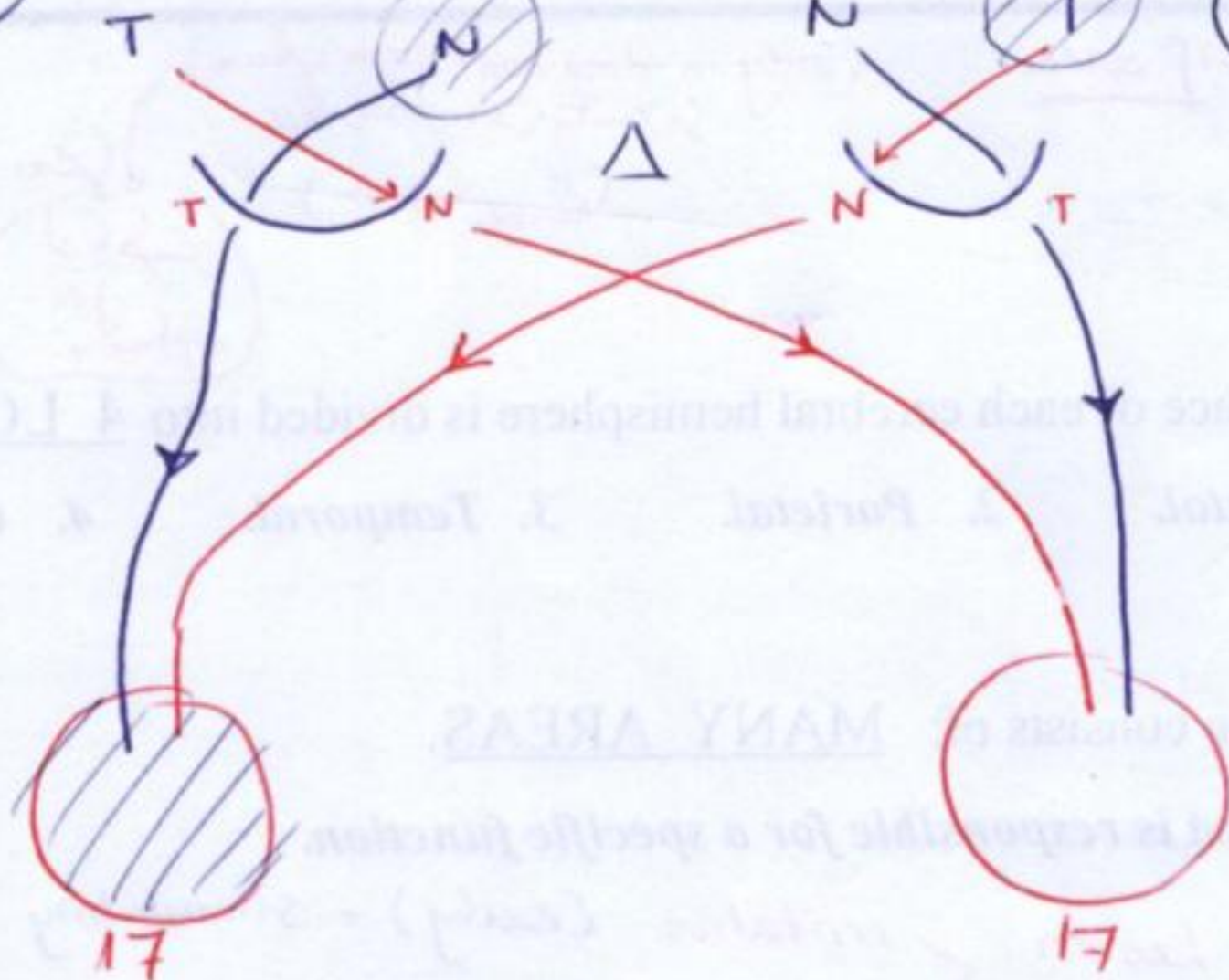
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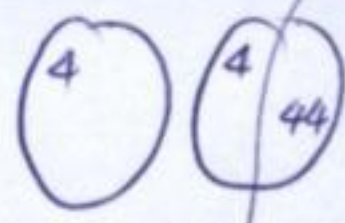
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R1



Formulation "44" aphasia
 articulation "4" # dysarthria



SPEECH DISORDERS

- Normal speech involves 2 stages:

1. **Formulation** whether Spoken or Written, This needs:

A) Sensory System:

1. Visual areas: 17, 18, 19, and 39.
2. Auditory areas: 41, 42 and 22.

B) Motor System:

1. Broca's area: 44.
2. Exner's area: 45.

C) Associative System:

- Supramarginal gyrus: 37.

Areas 17, 41, 42 are present in BOTH hemispheres.

Areas 18, 19, 39, 22, 44, 45, 37 are present in dominant hemisphere ONLY.

فكر
 كتب
 ربط

2. **Articulation** This needs:

A) UMN:

B) LMN:

- Cranial Nuclei (5, 7, 10, 12).
- Nerves. Cranial nerve
- NMJ.
- Muscles.

Pyramidal tracts. (4)

jaw
 lips, cheeks
 vocal cords
 tongue

orbicularis oris
 Retraction

Slurred

C) Cerebellum:

for co-ordination of the muscles of speech.

D) Extrapyramidal system:

for making the speech expressive.

فعلية

- Disorders of speech include:

APHASIA

Definition

- Difficulty or inability of the formulation of speech:
 - In the absence of lesions of the sense organs (e.g. Vision or Hearing).
 - In the absence of mental defect.

Types

1. Sensory Aphasia: “due to defect in comprehension”

a) Visual:

- Visual agnosia: (lesion in area 18, 19)
The patient sees objects but does not recognize them.
- Alexia: (lesion in area 39)
The patient cannot read because he does not understand the letters & numbers (word blindness).

b) Auditory:

- Auditory agnosia (lesion in area 22)
The patient hears sounds but does not understand them.

2. Motor Aphasia: “due to defect in expression”

a) Verbal aphasia: (lesion in Broca's area)

The patient cannot express himself in spoken words; (although he can understand visual & auditory stimuli).

b) Agraphia: (lesion in Exner's area)

The patient cannot express himself in written words (although he can understand letters & numbers).

Associative

3. Jargon's Aphasia (Word salad): “due to defect in association”

- It is due to lesion in the supramarginal gyrus (area 37).
- The patient can speak, **BUT**, the words are meaningless & not related.
- This condition is usually associated with apraxia.

لا يأتى سؤال أبداً
فقط Clinical
سببها
hemiplegia
اللي ياتى
aphasia

من يقول طلب
انكى انه يعرف يكتب

لازم

DYSARTHRIA

حالة صرّة

Definition

- Difficulty in articulation of speech (with normal formulation of speech).

Types

1. Slurred Speech:

UMNL
LMNL

ياكل الكلام
أو لسانه ثقيل

- Disturbance in the production of consonants especially:

- o Labials (e.g. B, M).
- o Dentals (e.g. D, T).

أما الحروف
a e i o u y

يظهروا بسهولة مع صوت

- Causes include:

a) Bilateral pyramidal tract lesion (Cortico-bulbar lesion), as in:

- o Pseudobulbar palsy.

b) LMNL of the cranial nerves concerned with speech, e.g.:

- o Nuclear: True bulbar palsy.
- o Nerve: Facial nerve palsy. P-Neuritis eg VII, X, XII
- o NMJ: Myasthenia.
- o Muscle: Myopathy (Facio-scapulo-humeral myopathy).

v. rare

face

shoulder

2. Staccato Speech:

كلام مقطّع

- The speech is explosive with separation of the syllables.

- Causes include:

- o Hereditary ataxias. Friedreich's
- o DS. Marfan's

Cerebellar lesions:

DCM قديما في

Friedreich's = CP

3. Scanning Speech:

ياكل الكلام
ومقطّع

(Slurred Staccato)

- It occurs also in: Cerebellar lesions.

+ Δ lesion

D.S.

Friedreich's
& Marfan's
e. path

Cerebellar & Δ
lesion

4. Monotonous Speech:

ونبرة واحدة

- The speech is expressionless & monotonous.

- Causes include:

- o Parkinsonism.

Extrapyramidal lesions:

B. G.

THE CRANIAL NERVES

1. OLFACTORY NERVE

Function:

- Sense of smell.

Lesion:

1. Anosmia:

"loss of sense of smell"

a) Bilateral anosmia:

causes:

- ENT causes, e.g. atrophic rhinitis.
- Congenital.

b) Unilateral anosmia:

causes:

- Traumatic: fracture cribriform plate (in fracture base of skull).
- Inflammatory: basal meningitis.
- Neoplastic: meningioma of the olfactory groove.

2. Olfactory Hallucinations:

"false perception of bad smell"

- It occurs in irritative lesions of the limbic system in the temporal lobe.

clinical

facial n. & its anatomy

① - facial n. & its anatomy

② Trigeminal

③ optic

اعلى منى
سوال لوجه
ركي خلاصه
خاتمة في
درس
blood supply
brain

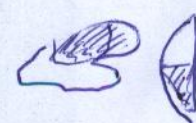
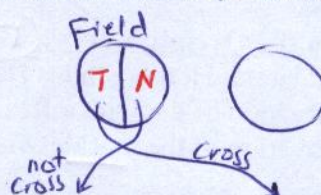
2. OPTIC NERVE

Pathway:

- From the retina, the nerve fibres converge to form the **optic nerve**.
- The **nasal fibres** of the nerve (which carry the temporal field of vision), decussate at the **optic chiasma** to pass in the optic tract of the **opposite side**.
- The **temporal fibres** of the nerve (which carry the nasal field of vision), pass in the **optic tract** of the **same side**.
- The fibres relay in the lateral geniculate body, where new fibres arise & pass in the posterior limb of the **internal capsule**. (Δ , Sensory, visual)
- The fibres then pass in the **optic radiation** (in the parietal & temporal lobes).
- The fibres of the optic radiation finally terminate in **area 17** of the **occipital lobe** (visual sensory area), where vision is perceived.
- The neighbouring **areas 18 and 19** (visual associative areas) are responsible for the recognition & recall of images.

Function:

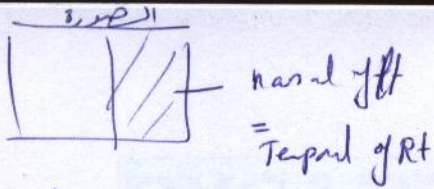
- Sense of vision.



upper fibres from lower 1/2 of opp. 1/2

lower fibres from upper 1/2 of opp. 1/2





والحيز السليم يتوقف

• **Lesion:** differs according to the site in the pathway:

1. **Lesion in the Optic Nerve:**

- Ipsilateral loss of vision.
- ✗ - Loss of direct & consensual light reflex.

direct
or
consensual } واحدة
تضع
لوحظ
III

2. **Lesion in the Optic Chiasma:**

- Bitemporal Hemianopia.
- ✗ - loss of direct & consensual light reflex

II اما حاليه حيز

3. **Lesion in the Optic Tract:**

- Contralateral Homonymous Hemianopia (bilateral half blindness).
- ✗ - loss of direct & consensual light reflex

direct
consensual
bip

4. **Lesion in the Optic Radiation (lower fibres):** temporal

- Upper quadrantic Contralateral Homonymous Hemianopia.
- ✓ - Preservation of the light reflex.



5. **Lesion in the Optic Radiation (upper fibres):** parietal

- Lower quadrantic Contralateral Homonymous Hemianopia.
- ✓ - Preservation of the light reflex.



In complete lesion of the Optic Radiation, there will be:

- Contralateral Homonymous Hemianopia (bilateral half blindness).
- Preservation of the light reflex.

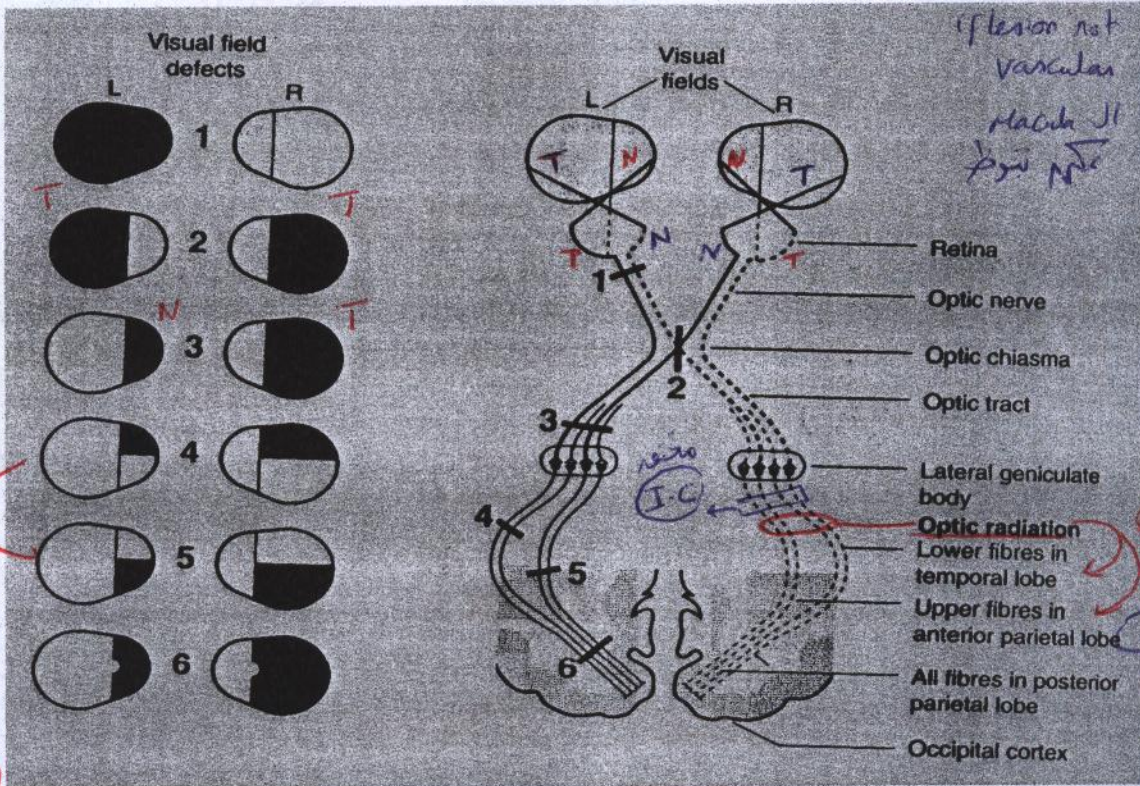
6. **Lesion in the Occipital Lobe:** (VT)

- Contralateral Homonymous Hemianopia (bilateral half blindness).
- ✓ - Preservation of the light reflex.
- Preservation of the macular vision (due to double blood supply).

other



optic tract
I.C. 5
3 1



LIGHT REFLEX

Definition:

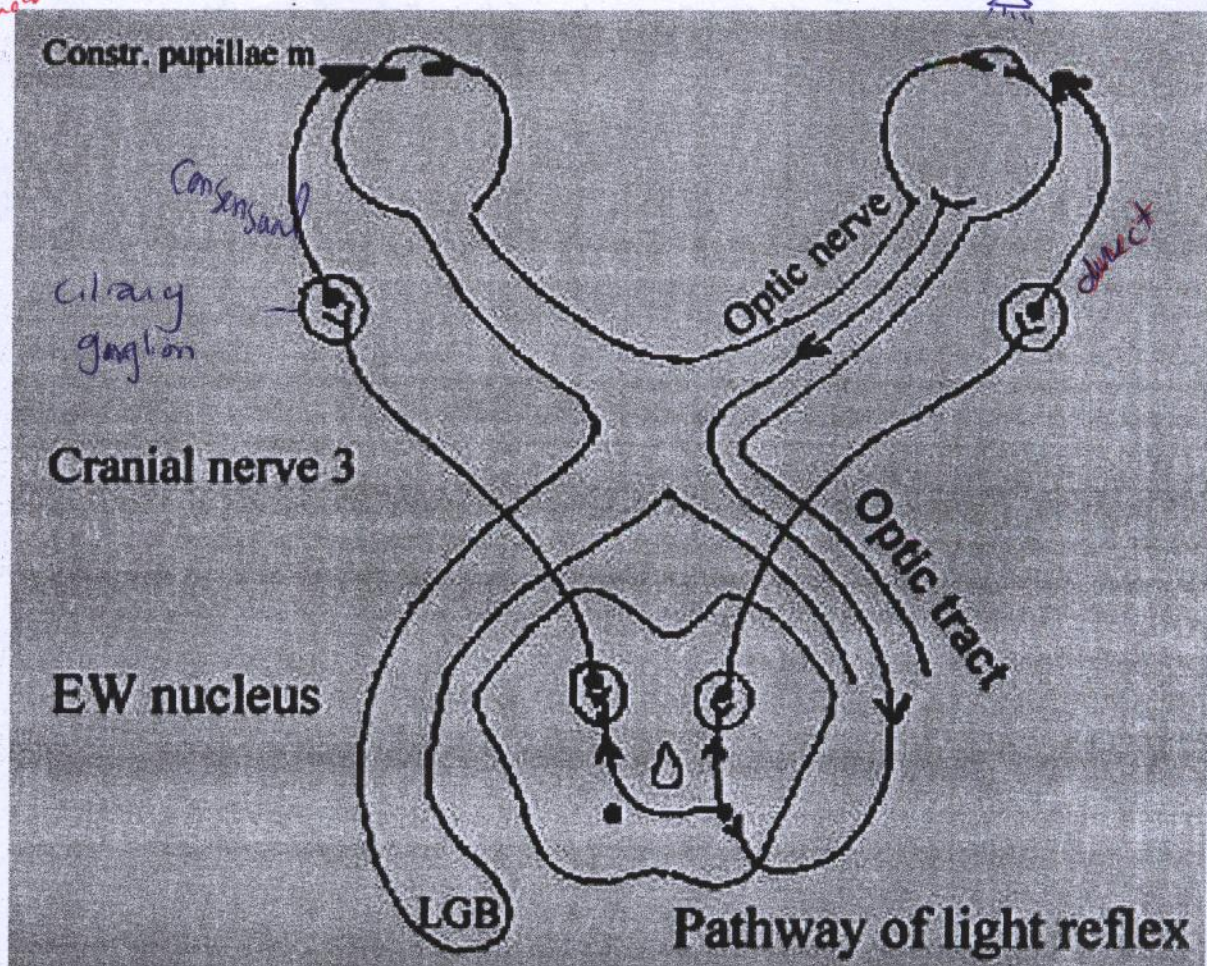
- Exposure of one eye to bright light results in :
 1. Constriction of the same side (direct reflex).
 2. Constriction of the opposite side (consensual reflex).

Stimulus: light
 APF: II
 Centre: MB (EWN)
 Eff: III
 MS: constriction

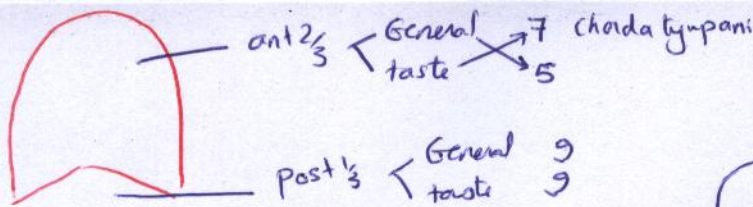
Pathway:

- Impulses pass in the optic nerve, chiasma & then optic tract.
- Fibres do not reach the lateral geniculate body, but pass to the Edinger-Westphal nuclei (EW nuclei) of both sides in the midbrain.
- Fibres then pass from the EW nuclei of both sides to the oculomotor nerves.
- Fibres finally terminate in the constrictor pupillae muscles of both eyes.

Part of
 Nucleus
 III
 oculomotor



Oral
عمالي
الفم



Clinical
+
Sp

29

5. TRIGEMINAL NERVE

Function:

* Mixed $\frac{M}{S}$ pair

- 1 Anatomy
- 2 Lesions
- 3 D.D. of facial pain

1- The Sensory Division:

- It conducts sensations from the face (except the angle of the mandible, supplied by C2), the anterior 2/3 s of the tongue & the buccal cavity.
- It is formed of 3 branches: ophthalmic, maxillary, and mandibular.
- Pathway:

a) Fibres carrying pain & temperature sensations relay in the spinal sensory nucleus in the medulla on the same side, where:

- The upper face is represented in the lower part of the nucleus & vice versa.
- The outer face is represented in the inner part of the nucleus & vice versa.

b) Fibres carrying deep sensations relay in the mesencephalic nucleus in the midbrain on the same side.

c) Fibres carrying touch relay in the main sensory nucleus in the pons on the same side.

- From these 3 nuclei, new fibres cross to the opposite side, then ascend with the medial & lateral lemnisci, to reach the thalamus.

- From the thalamus, fibres carrying cortical sensations of the face ascend to end in the cortical sensory area in the parietal lobe. passing thru Internal Capsule

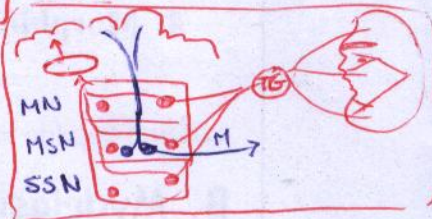
2- The Motor Division:

- It supplies the muscles of mastication.
- It arises from the motor nucleus in the pons.

Temporalis
Masseter
Pterygoids

same side

deep
touch
Pain temp



Lesion:

1. Sensory affection:

P.N. = a) Peripheral lesion:

- Loss of sensations on the same side of the face (sparing the angle of the mandible).
- Loss of general sensations over the anterior 2/3 s of the tongue, (in lesions of the mandibular division).

Brain stem = b) Central lesion: (mainly affects the spinal sensory nucleus in the medulla):

- Ipsilateral dissociated sensory loss of the face, i.e. lost pain & temperature with preservation of touch & deep sensations.
- The clinical picture varies according to the site of the lesion:

- Lesion starting from above (lower pontine tumour): affects the lower part of face.
- Lesion starting from below (upper cervical lesions): affects the upper part of face.
- Lesion starting from periphery (Tabes dorsalis): affects the central part of face.
- Lesion starting from midline (syringobulbia): affects the outer part of face.

Brain stem tumor

degenerative

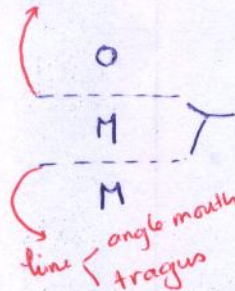
as syringomyelia
- sp cord
(paraplegia)

etio: long standing untreated syphilis

• patho: degeneration & chronic inflamm.
• site: post root (dorsal) White Knight Love
From outwards inwards

29

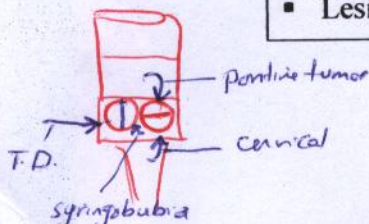
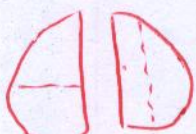
angle eye
line tragus



superficial
deep

if lesion

- thalamus } opp side
- nucleus } same side
- C.N. }



Oral

directions of dentate

- Tongue 12 & jaw 5
same side (diseased side)
- angle of mouth 7 & uvula 9, 10
opposite (healthy)

P.N.

2. Motor affection:

- Weakness of the muscles of mastication on the same side of the lesion.
- Deviation of the jaw to the affected side due to the unopposed action of the pterygoid muscles of the healthy side.

temporalis
masseter
pterygoids

30

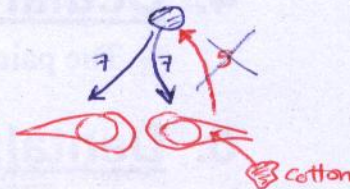
3. Reflex affection:

- Ipsilateral loss of the corneal reflex (afferent 5, efferent 7).
- Ipsilateral loss of the palatal reflex (afferent 5, efferent 10).
- Appearance of jaw reflex (afferent 5, efferent 5):

in cases of bilateral UMNL above the pons.

sudden forcible closure of jaw

= Bilat loss of blinking



DD OF FACIAL PAIN

1. Trigeminal neuralgia:

Clinical picture: etio idiopathic irritation of V roots

- Severe attacks of unilateral pain along one or more of the sensory branches of the trigeminal nerve, especially the maxillary & mandibular.
- In between the attacks the patient is completely free.
- It usually affects middle age, more commonly females.

no complications

Etiology:

- The exact cause is UNKNOWN, but there are predisposing factors:

- Root compression by a tumour in the cerebello-pontine angle.
- Trigeminal neuralgia is common in: **DS & DM.**

The attacks are precipitated by movements of the jaw as laughing & mastication.

Treatment:

- Medical:

- Carbamazepine (Tegretol): 500 – 1000 mg / day orally.
- Analgesics.

- Surgical:

- e.g. Section of the affected sensory root.

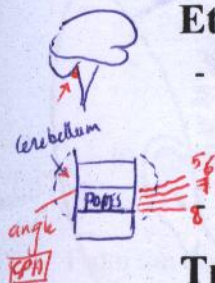
2. Post-herpetic neuralgia:

- The pain usually affects the ophthalmic division.
- The pain is followed by herpetic skin eruption.

Severe Facial burning pain followed by after 3-4d by vesicular skin rash

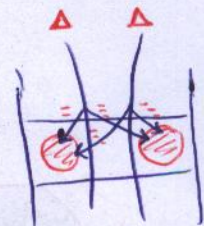
especially ophthalmic in an immuno compromised patient

White Knight Love



hyperreflexia up to clonus

pathological reflex



above pons: as motor nucleus is in pons
UMNL & lesion in pons
ظاهر التأثير
below level

Bilat: لانه لو واحد پس
هو الى انتحرب - لا يزال
inhibition from other Δ

peripheral neuritis (PN)
early: pain & burning & late: lost sense.

early

زيادة احساس
واخذ الدواء
وهو يترافق وقتها
علامه الصرع
رؤيت تطننه
30

3. Sinusitis:

- The pain is worse in the morning, with tenderness over the involved sinus.

4. Ocular (glaucoma):

- The pain is behind the eye, with associated visual symptoms.

5. Dental:

- The pain is around the mouth.

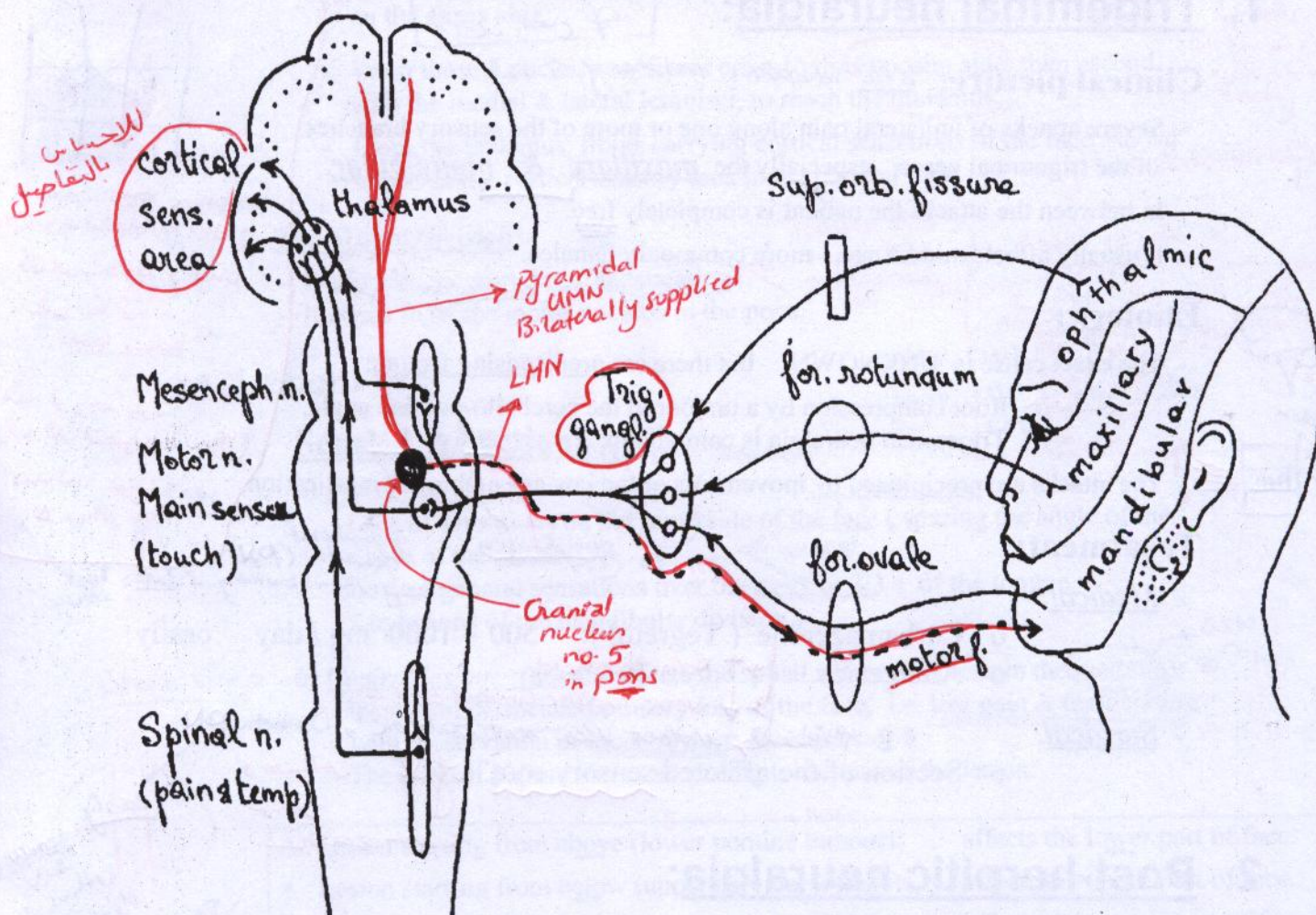
6. Cluster headache:

- Refer to the chapter of "Migraine".

retention of secretions

أسر ← ب

PATHWAY OF THE TRIGEMINAL NERVE



7. FACIAL NERVE

- **Function:**

The facial nerve is a mixed nerve, containing: motor, sensory and autonomic fibres.

The **motor** part: supplies the **muscles of expression** of the face as well as 4 other muscles:

- Platysma.
 - Posterior belly of the digastric muscle.
 - Stapedius.
 - Stylohyoid.
- in Middle ear to dampen the sound (if high) if Stapedius*

The **sensory** part: receives taste sensations from the anterior 2/3 s of the tongue.

The autonomic part: supplies the lacrimal gland as well as the submandibular and sublingual salivary glands.

→ only
مفیدی ای
فانی نقل ال

to dampen
if stapedius
↓
new caput
↑ up the
↑ sensitivity to
sound
hyperacusis

- **Anatomy:**

1. Anatomy of the motor part:

“ See figure ”

The **motor nucleus** of the facial nerve lies in the **pons**, near the 6th nerve nucleus. It is controlled by 2 types of supranuclear fibres:

a) Pyramidal Fibres:

- The upper part of the nucleus is supplied from both pyramidal tracts. (corticobulbar)
- The lower part of the nucleus is supplied from the opposite pyramidal tract only. (corticobulbar)

b) Extra-pyramidal Fibres:

- For emotional & associated movements.

From the nucleus, the motor fibres form a loop around the 6th nerve nucleus, then pass laterally to emerge at the lower part of the pons.

The nerve runs laterally between the 6th and 8th cranial nerves, in the subarachnoid space of the **cerebellopontine angle** to enter, through the **internal auditory meatus IAM**, into the facial canal.

(5) In the **facial canal**, the motor part of the facial nerve becomes adherent to its sensory and autonomic parts. It gives the **nerve to Stapedius muscle**.

Finally, the facial nerve leaves the canal through the **stylomastoid foramen SF**, passes through the **parotid gland** to divide into its terminal branches to innervate the muscles of the face.

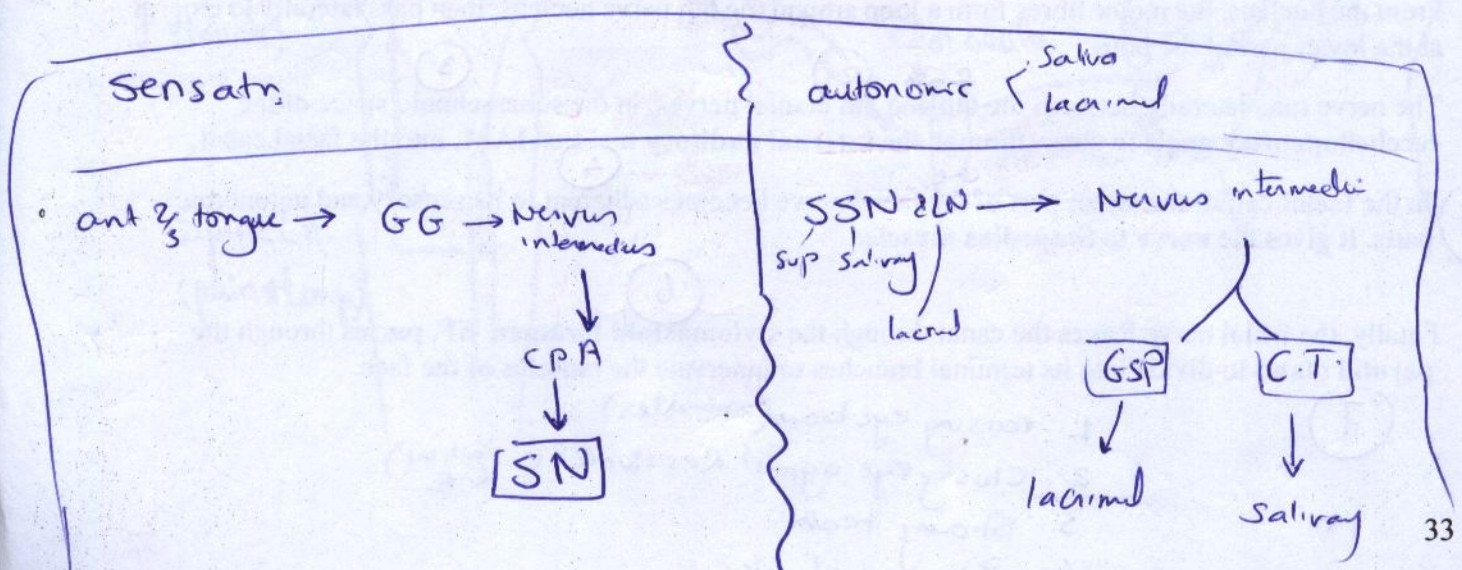
1. raising eyebrow (wrinkles)
2. closing eye against Resistance (عَلْيَ ١١)
3. Showing teeth
4. Blowing cheek

2. Anatomy of the sensory and autonomic parts: "See figure"

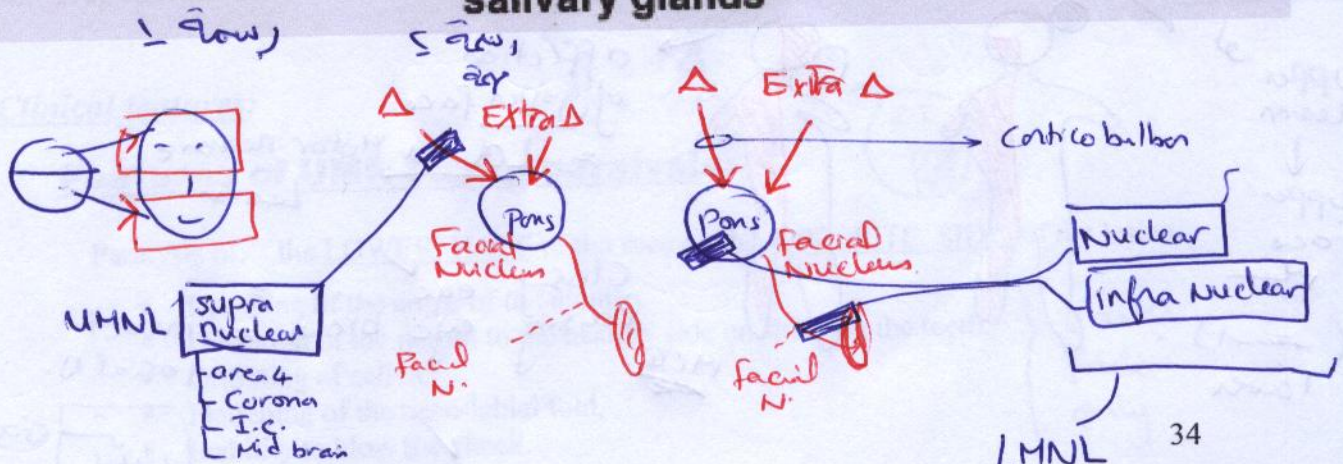
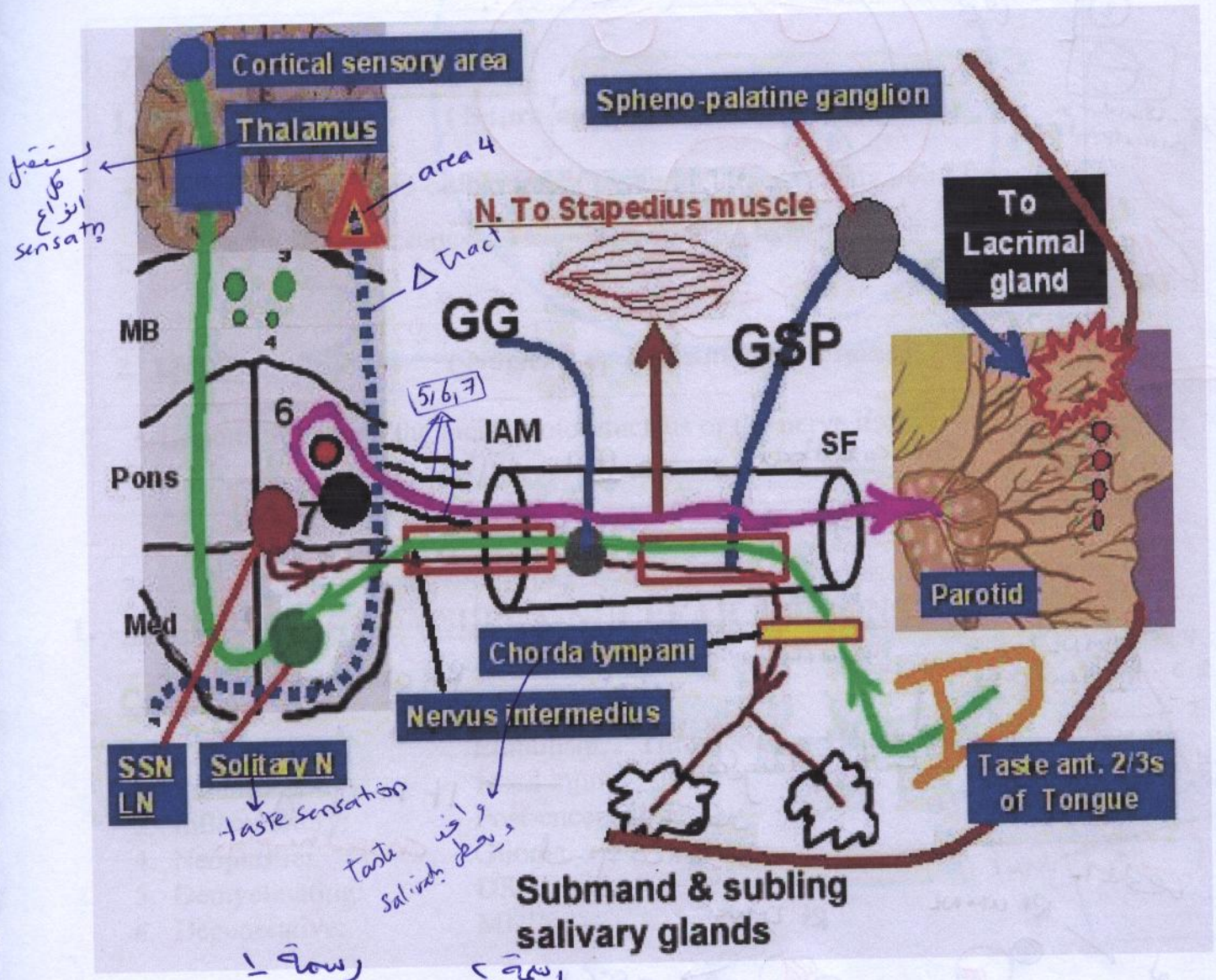
In the facial canal lies the **geniculate ganglion**.

This ganglion gives 2 branches, a **peripheral branch** & a **central branch**.

- a. The **peripheral branch**: runs laterally and divides into the greater superficial petrosal nerve and the chorda tympani.
- The **greater superficial petrosal GSP** nerve passes forwards to relay in the sphenopalatine ganglion where a new set of fibres gives autonomic supply to the lacrimal gland.
 - The **chorda tympani** leaves the facial nerve before the stylomastoid foramen, to supply the submaxillary and sublingual salivary glands and to carry taste sensations from the anterior 2/3s of the tongue.
- b. The **central branch**: passes centrally, joins the motor part of the nerve, then enters the cranial cavity through the internal auditory meatus as the **nervus intermedius**.
- This nervus intermedius then enters the brain stem to terminate in the **solitary nucleus** in the medulla.
 - A new set of fibres passes from the nucleus to the opposite side and runs upwards to terminate in the lower part of the **cortical sensory area**, where taste sensation from the anterior 2/3s of the tongue is perceived.

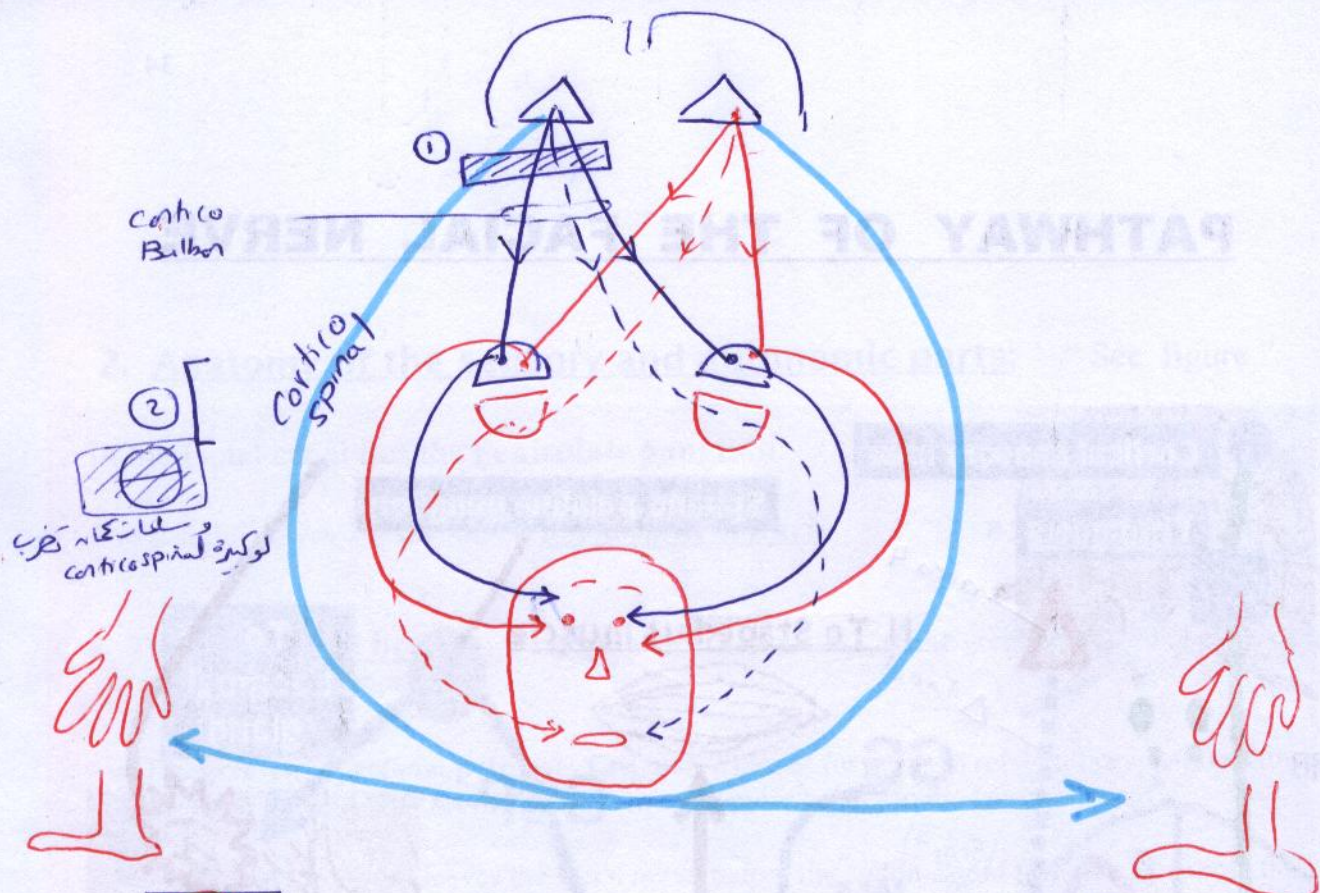


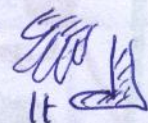
PATHWAY OF THE FACIAL NERVE



only voluntary affects
 as only Δ is affected
 while Extra Δ intact emotion associated ✓

LMNL 34
 vol. & Δ emotions associated
 Both affected
 as both Δ & Δ X

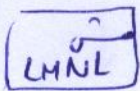


① **RT UMN** { Corticobulbar → Lt lower face only 
 Corticospinal → Lt hemiplegia → 

② **RT LMN nuclear pons** { nucleus → Rt all face 
 + if large lesion → Lt hemiplegia
Crossed hemiplegia

المخيف
 RT UMN
 upper lesion
 ↓
 upper face
 (مخيف)
 lower



RT LMN
 affection of upper face
 = Lower Motor neurone Lesion
 closing eye
 Raising eye brows = intact facial N.
 LMN 



* علامه بجایه ای سوال. ابدأ اندک شوت فشاره! مهموم

لو سیر ← UMNL عل الناحیه العکس

لو بايظ يحن من مائل عينه { ← LMNL عل نفس الناحیه
ارفضيف كرمه فونه

35

• Lesions of The Facial Nerve

The lesion may be:

1. UMNL: (Supra-nuclear lesions):

- Lesions of the cortico-bulbar tract (pyramidal tract) at any point from:
The motor area in the cortex → to the facial nucleus in the pons.

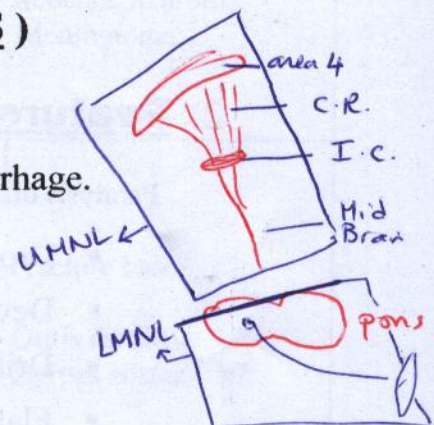
2. LMNL: (Nuclear & infra-nuclear lesions):

- Lesions that affect the facial motor nucleus or the nerve itself.

1. UMNL: (SUPRA-NUCLEAR LESIONS)

- Causes:

- | | |
|-------------------|-----------------------------------|
| 1. Vascular: | Embolism, Thrombosis, Hemorrhage. |
| 2. Traumatic: | Head injury. |
| 3. Inflammatory: | Post-encephalitic. |
| 4. Neoplastic: | Glioma. |
| 5. Demyelinating: | DS. |
| 6. Degenerative: | MND. |



- Clinical features:

Features of UMN Facial paralysis:

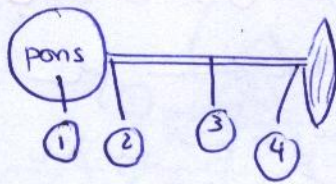
Paralysis of: the LOWER HALF of the face on the OPPOSITE SIDE of the lesion:

- Dropping of the angle of the mouth.
- Deviation of the mouth to the healthy side on showing the teeth.
- Dribbling of saliva.
- Flattening of the naso-labial fold.
- Inability to blow the cheek.
- Accumulation of food behind the cheek.

} Lower Face only



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2. LMNL: (NUCLEAR & INFRA-NUCLEAR LESIONS)

- Causes:

1. Pontine Lesions.

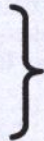


Nuclear Lesions

2. Cerebello-Pontine Angle

3. Facial Canal Lesions.

4. Extracranial Lesions.



Infra-nuclear Lesions

(Internal auditory meatus) — IAH ~ Canal يدخل •
(stylo mastoid Foramen) — SMF ~ skull ~ ورم •

- Clinical features:

1. Features according to the level of the lesion (see later: *).

2. Features of LMN facial paralysis:



Paralysis of: ALL MUSCLES of the face on the SAME SIDE of the lesion:

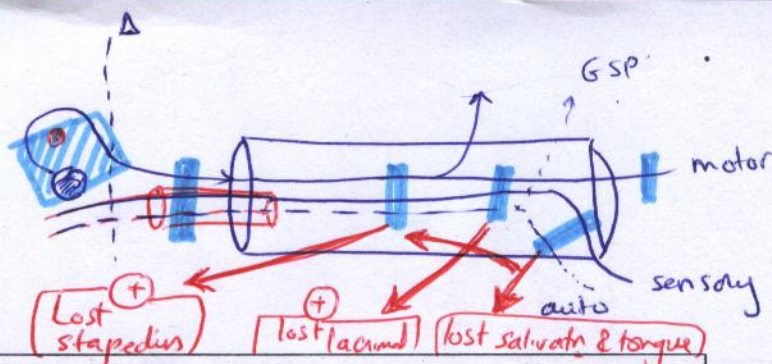
- Drooping of the angle of the mouth.
- Deviation of the mouth to the healthy side on showing the teeth.
- Dribbling of saliva.
- Flattening of the naso-labial fold.
- Inability to blow the cheek.
- Accumulation of food behind the cheek.

lower 1/2

PLUS

- Inability to raise the eye brows.
- Inability to close the eyes.
- Absence of wrinkles of the forehead on looking upwards.

upper 1/2



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*

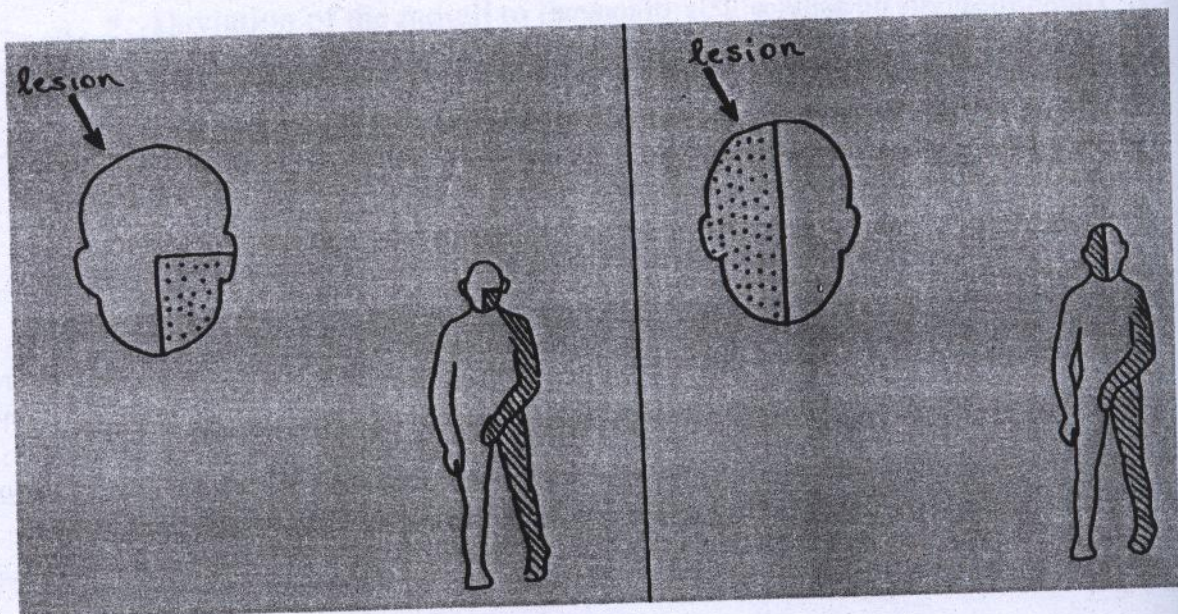
LOCALISATION OF THE SITE OF LMNL

SITE OF LESION	CAUSES
Pontine Lesions: (Nuclear Lesions) 1. Paralysis of facial muscles. <i>LMN same side</i> 2. <u>No</u> impairment of taste sensation. 3. <u>No</u> impairment of salivation nor lacrimation. 4. May be other <u>LMN</u> cranial palsies on the <u>same</u> side or hemiplegia on the opposite side. 5. <u>Hyperacusis</u> <i>لو كبرى ضربت corticospinal (Δ)</i> <i>الاحقر البنى</i> <i>stapedius ضربت</i>	<i>Carotid</i> <i>Brainstem</i> 1. Vascular: - <u>Vetebro-basilar</u> insufficiency. - Millard-Gubler syndrome. 2. Infective: - Encephalitis - Poliomyelitis. 3. Neoplastic: - Glioma. 4. Demyelinating: - DS.
Cerebello-Pontine Angle Lesion: (ALL) 1. Paralysis of facial muscles. 2. <u>Diminished</u> taste on anterior 2/3s of tongue. 3. <u>Diminished</u> salivary & lacrimal secretions. 4. Associated Cr. 5, 6 & 8 palsies on same side. 5. Ipsilateral cerebellar ataxia. <i>(as cerebellar fibers not cross)</i> 6. <u>Hyperacusis</u>	1. Infective: - Basal meningitis. 2. Neoplastic: - Acoustic neuroma. - Meningioma.
Facial Canal Lesion: (If) 1. Paralysis of facial muscles. 2. Diminished taste on anterior 2/3s of tongue and diminished salivation if chorda tympani is involved. 3. Diminished lacrimation if the greater superficial petrosal nerve is involved. 4. Hyperacusis if the nerve to Stapedius is involved.	1. Traumatic: - Fracture base. 2. Infective: - Otitis media. - Herpes zoster. 3. Neoplastic: - Facial neuroma. 4. Bell's palsy.
Extracranial Lesions: (After its exit from the stylomastoid foramen): Paralysis of facial muscles <u>only</u> .	1. Neuropathy: - Diabetic - Leprotic. 2. Myopathy: - Facioscapulohumeral. <i>skinned speed</i> 3. Myasthenia. 4. Invasion by a tumour: e.g. <u>from parotid</u> . 5. Injury to the nerve during surgery.

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Differentiation Between UMNL & LMNL of the Facial Paralysis

UMNL	LMNL
1. Affects the Pyramidal tract above the Facial Nucleus.	1. Affects the Facial Motor Nucleus, or, the Nerve itself.
2. Paralysis of the muscles of <u>LOWER HALF</u> of the face on the <u>OPPOSITE SIDE</u> of the lesion (supplied from the opposite pyramidal only).	2. Paralysis of <u>ALL MUSCLES</u> of the face (upper & lower halves), on the <u>SAME SIDE</u> of the lesion).
3. Paralysis involves the voluntary movement, <u>BUT</u> : spares the emotional & associated movements (supplied from the extra-pyramidal fibres).	3. Paralysis involves the voluntary, emotional and associated movements.
4. Paralysis is associated with Hypertonia and Hyperreflexia.	4. Paralysis is associated with Hypotonia and Hyporeflexia.
5. There is associated Hemiplegia on the <u>SAME SIDE</u> of the facial paralysis.	5. من لایه If there is Hemiplegia, it is on the <u>OPPOSITE SIDE</u> of the facial paralysis (crossed Hemiplegia).



بعضی SQ

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Bell's Palsy = Cranial VII LMNL

DEFINITION

- It is an acute LMN paralysis of the face, due to a non-suppurative inflammation & oedema of the facial nerve in the FACIAL CANAL near the stylomastoid foramen.
- It is usually unilateral, may be recurrent & sometimes runs in families.

ETIOLOGY

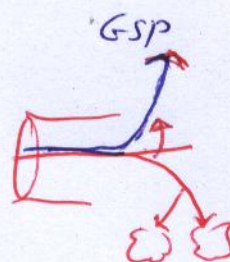
- UNKNOWN: Many causes have been suggested:
 1. Air drafts: Exposure to air drafts may lead to inflammation, oedema, & compression of the nerve at the stylomastoid foramen.
 2. Viral infection: It may be secondary to a neurotropic virus (Herpes zoster), Herpes simplex.
 3. Autoimmune: There is evidence of high levels of immunoglobulins in the patient's serum.

CLINICAL PICTURE

1. PAIN:
 - The entire course of the disease may be PAINLESS, OR,
 - The disease may also present with acute PAIN behind the ipsilateral ear.
2. PARALYSIS: "one or two days after the pain"
 - Complete paralysis of the facial muscles on the affected side of LMN features, (features of FACIAL CANAL lesion).

COMPLICATIONS

1. CROCODILE TEARS:
 - Excessive tears: flow from the affected eye during eating.
 - Explanation: the regenerating fibres to the submandibular salivary glands become misdirected and reach the lacrymal gland.
2. CORNEAL ULCERS.
3. INCOMPLETE RECOVERY:
 - This may result in residual facial asymmetry.



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TREATMENT

۴ دیکارے

1. REASSURANCE.

دکتور
نفسیہ

2. PHYSIOTHERAPY:

دکتور
علاج
فیزی

- Massage of the facial muscles.
- Infrared irradiation to the face.
- Galvanic stimulation to the facial muscles.

3. MEDICAL TREATMENT:

دکتور
باجنہ

- Corticosteroids: Oral prednisolone (1 mg / Kg / day): to ↓ the inflammation & oedema.
- Corneal protection: Eye ointment during sleep, sunglasses during daytime.

مشاورت دکتور

4. SURGICAL TREATMENT:

- For Facial nerve palsy: Decompression of the facial nerve or Facial nerve grafting.
- For Crocodile tears: Section of the regenerating fibres.
- For Residual asymmetry: Plastic surgery.

یقیناً

لو لم وقف عضوها
في

PROGNOSIS

القادره

- Most patients (80%) recover in few weeks.
- The remaining (20%) need surgical interference.

لا

تجديد

CEREBRO-VASCULAR DISEASE

Diseases of the **BRAIN VESSELS** include:

1. **STROKE.**

2. **TRANSIENT ISCHEMIC ATTACKS (TIAs).**

STROKE

DEFINITION

• **ACUTE INJURY** to the brain due to a **VASCULAR** problem.

• It produces neurologic deficits: = CIP

- **Acute in onset.** or v. **acute (sudden)** [never gradual]
- **Persistent for more than 24 hours.**

ETIOLOGY

1. **Ischemic stroke:**

"The most common cause"

85 %

OCCLUSION of a blood vessel to a part of the brain → Ischemia.

- **Thrombosis** on top of cerebral atherosclerosis:
- **Embolism:**

Acute occlusion.

Sudden occlusion.

2. **Hemorrhagic stroke:**

"Less common cause"

15 %

RUPTURE of a blood vessel in a part of the brain → Bleeding.

1. **Thrombosis:**

"**clot in situ** resulting in cerebral infarction"

1. **Vessel wall diseases:**

- Cerebral atherosclerosis:
- Vasculitis:

THE MOST IMPORTANT.

PAN & SLE.

2. **Blood diseases causing hyperviscosity:**

- Polycythemia (↑ RBCs).
- Leukemia (↑ WBCs).
- Thrombocytosis (↑ Platelets).

3. **Circulation diseases:**

SLOW CIRCULATION → THROMBOSIS.

- Heart failure.
- Systemic hypotension, e.g. Dehydration & shock.

causes **contact** → ++ platelets (vWF)
++ intrinsic → HMWK White Knight Love
pre kallikrein

الأسف
MS + AF

2. Embolism:

جاء من مكان آخر

sudden (seconds)

45

"wandering clot resulting in cerebral infarction"

MS + AF =
35% - 40%
↑ in emboli

The source of the embolus may be:

1. Heart:

"The most common source"

eg ① - MS with AF,
② - MVP
③ - CM

② Vegetations of SBE,
↳ embolic
hemiplegia

③ Mural thrombus in AMI.

"Late complication"
↳ stroke is atypical presentation of CAB

2. Distal vessels:

Arterial: Detached atheromatous plaque from carotid vessels or vertebrobasilar A.

Venous: DVT → detached thrombus → paradoxical embolism through VSD or ASD.
↳ reversed shunt

لوقية القلب
سوء
جدار
أخرى

3. Hemorrhage:

Types of Intracranial Hemorrhage:

1. Intracerebral:

- The bleeding is in the brain substance & may leak into the ventricles.
- This is very serious as the blood may compress vital centres. [mainly Brain stem]
- The commonest artery causing intracerebral hemorrhage is the "lenticulo-striate branch of the middle cerebral artery" mainly Medulla

2. Subarachnoid:

Bleeding is in the subarachnoid space.

3. Subdural or extradural:

Bleeding often forms a hematoma.

Causes of Intracranial Hemorrhage:

1. Hypertension:

The commonest cause of:

Intracerebral haemorrhage.

2. Rupture of an intracranial aneurysm, or A-V malformation:

The commonest cause of:

Subarachnoid haemorrhage.

3. Trauma to the head:

The commonest cause of:

Subdural haematoma.
↳ Extradural

4. Hemorrhagic blood diseases:

Purpura & Hemophilia.

5. Anticoagulants: if overdose (iatrogenic)

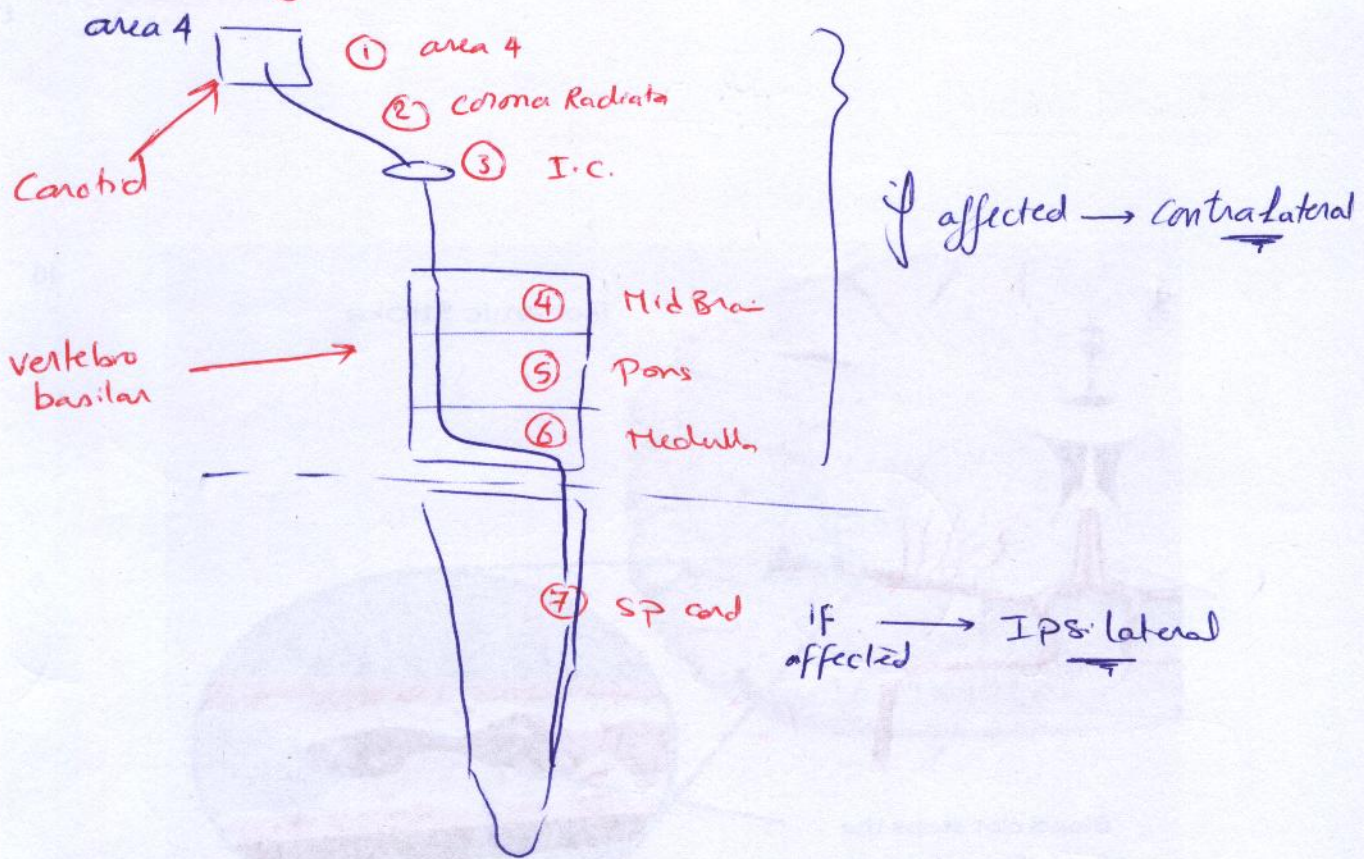
↳ if indicated for life must follow up:

• if c heparin → by APTT
• if c oral anti coag. → by PT (INR)

White Knight Love

45

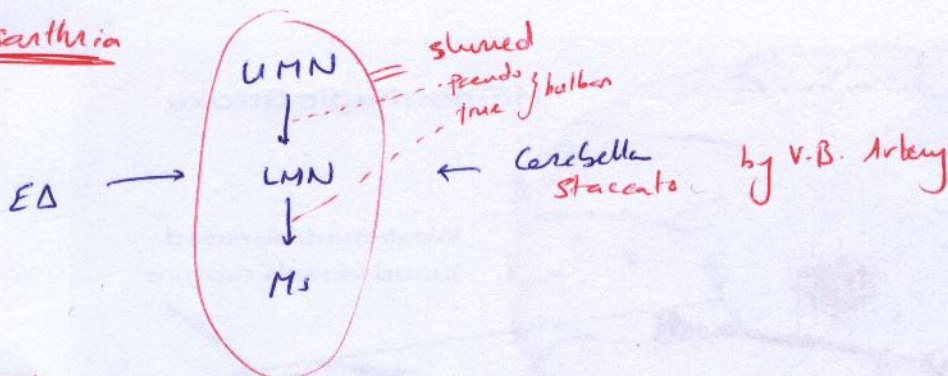
Hemiplegia



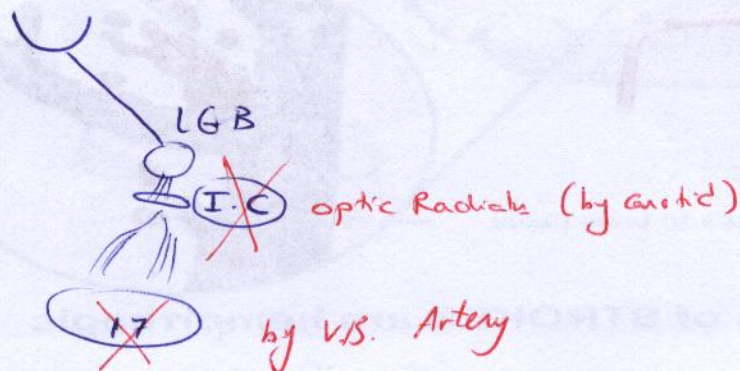
Aphasia

44 by carotid
18, 19

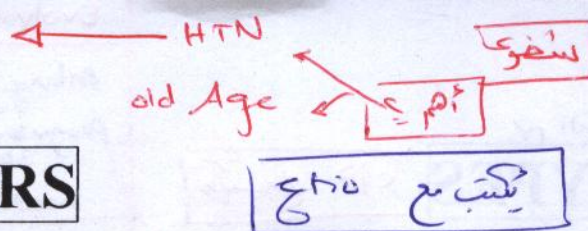
Dysarthria



Vision



esp. Systolic HTN



47

یہ صنفی heart

نہ رات Heart diseases

AF

RISK FACTORS

1. Non – modifiable:

- Age: old. (marked with a red star)
- Sex: male : female = 3 : 1.
- Type A personality : nervous, intellectual.
- Genetic factors: positive family history.

2. Modifiable:

a) High risk:

- Heart diseases: (marked with a red star) especially valvular heart diseases & AF.
- Hyperlipidemia: (marked with a red star) ↑ total cholesterol, ↑ LDL; ↓ HDL.
- **HYPERTENSION:** (marked with a red star) causes endothelial damage.
- Diabetes mellitus.
- Cigarette smoking.

b) Less risk:

- Obesity.
- Diet: rich in saturated fat & cholesterol.
- Physical inactivity.
- Psychological stress.
- Hyperuricemia.
- Homocysteinemia. (marked with a red star) # : Folic Acid
- Heavy alcohol intake.
- CCPs.

CLINICAL PICTURE

- Permanent neurologic deficits commonly produced by a stroke include:

1. HEMIPLEGIA. (marked with a red star) hemiplegia
2. HEMIANESTHESIA. (marked with a red star) spinal (as not stroke) Contra lateral
3. Speech problems: Aphasia or Dysarthria.
4. Vision problems: especially field defects.
5. Ataxia: Lack of co-ordination or Lack of balance.
6. Cranial nerve paralysis. (marked with a red star) in Brain stem by VB Artery

Ipsi lat. as cerebellum doesn't cross.

White Knight Love

CLINICAL TYPES

انكسب مع C/P

Evolving	infarct course	completed
enlarging	infarct course	stable
progressive 24-48	Anticoag.	stable
needed		not needed

1. Stroke in evolution:

(evolving stroke)

An enlarging brain infarct with neurologic deficits that worsen over 24 to 48 h.

2. Completed stroke:

A stable brain infarct with neurologic deficits that signify STABLE injury.

INVESTIGATIONS

I. BRAIN IMAGING

a) CT scan

1. Ischemic strokes:

- Infarctions may not appear until 24 hours after presentation.
- Small infarctions may not appear at all. → MRI

2. Hemorrhagic strokes:

- Bleeding appears immediately when it happens (even very small lesions).
- Therefore: CT scan can help to rule out a hemorrhagic stroke.

Good +ve & Good -ve 100%

b) MRI

It provides a more detailed and sensitive picture of the brain.

II. OTHER IMAGING

a) CARDIAC IMAGING:

b) VASCULAR IMAGING:

Chest X - Ray, ECG, Echocardiography.

DOPPLER ultrasonic imaging.

Atherosclerosis - distal vessels
Carotid V.B.

III. LABORATORY TESTS

a) CBC.

b) PT & APTT.

c) Risk factors:

e.g. blood glucose, lipid profile, uric acid, homocysteine.

high risk

low risk

TREATMENT

G
symp.
specific

I. GENERAL CARE:

"For: Hemiplegic patient or a Comatose patient"

"Stroke Unit"

emergency oral

1. Skin: (Bed sores)

- Frequent change of the patient's position (every 2 hours), and of the bed sheets.
- Frequent wash of the back & pressure points by alcohol followed by powder.

2. Swallowing & nutrition:

- Tube feeding. & IV fluids.

9, 10, 12 (bulbar palsy)

3. Balance of fluids:

- Controlled IV fluids.

Urine output
CVP

4. Breathing:

(Acute pulm. Edema)

- Suction of secretions.
- Oxygen inhalation.

5. Bladder:

- Catheterisation & urinary antiseptics.

6. Bowels:

- Daily enema.

(Const.)

GRF:

edema in brain of stroke pt?

① Injury → cytokines
↓
++ caps permeability

② Damage of BBB
(water tight endothelium)

Leads to enter of plasma fluid & ptn

II. SYMPTOMATIC:

1. EARLY:

- Cerebral dehydrating agents (Mannitol or Corticosteroids): to ↓ brain oedema.
- H₂ receptor antagonists (Ranitidine): to guard against stress ulcer.

edema
by osmotic diuresis

Local edema
very early

concentrated

2. LATE:

PHYSIOTHERAPY.

3. ALL through:

Vitamin and tonics.

ایسی علاج چواری

سٹریسز اولر ایسٹوپی

ایسٹوپی

III. SPECIFIC:

"TTT of the cause"

دوره ایست

دوره ایست
HI
Pulm Embolism

A) Thrombosis:

* 1. Control of Blood Pressure. eg 1 or 2 drugs

2. Thrombolytic therapy:

using **IV tPA** may be of benefit.

anticoag.
SK or UK

3. Antiplatelets:

because platelet aggregation is increased after thrombosis:

- **Aspirin (low dose):** 75 – 300 mg / day (single dose).
- **Dipyridamole:** 75 mg twice daily.
- **Ticlopidine:** 250 mg twice daily.

4. Anticoagulants:

They are not used in all cases:

total

Indications:

1. Stroke in evolution: i.e. gradual progressive weakness (over days) denoting gradual & progressive thrombus formation.
2. Recurrent TIAs. ^{H/O} Stroke
3. Embolic hemiparesis: esp. in cardiac cases to prevent recurrent embolisation.

Contraindications:

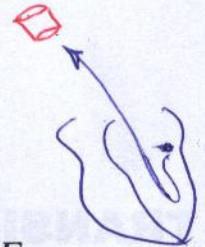
1. Haemorrhagic infarction; it is excluded by using the CT scan.
2. Haemorrhagic blood diseases.
2. Hepatic & Renal failure. (coated platelets & clotting factors by toxin)
3. Subacute bacterial endocarditis (SBE).

5. Other drugs:

- Trivastal or Trental:

may ↑ the cerebral blood flow.

death (if on anti-coag.)



B) Embolism:

1. Treatment of the source of emboli: e.g. MS with AF.
2. Anticoagulants: esp. in cardiac cases to prevent recurrent embolisation.

ICU
 ↓ BP. in thr.
 DBP not < 100
 Digoxide, Na Nitropruside
 & MD, ...

General measures.

C) Hemorrhage:

* 1. Control of Blood Pressure:

- Refer to: Hypertensive emergencies in Cardiology.

2. Antifibrinolytic drugs:

e.g. amino caproic acid

- They delay clot lysis & thus prevent rebleeding, However:
- They may ↑ cerebral ischemia. due to VC in surrounding B.V.

3. Surgery:

- Evacuation of the haematoma provided it is (not intraventricular.)

accessible

برة
 اما لو جره بينه
 ادخل

Injury by ↑ BP

Bleed → Clot

قفزة
 توقف النزف



فبرينوليتيك
 فبرينوليتيك

Plasminogen

plasmin →

الحلقة

Amino caproic acid

فيحافظ على الجلطة

Strokes either preceded by TIA or not

دلیل کثیر، البته علت Atherosclerosis و Ppt. factor. هفتاد و نه درصد الناس که تھقل (over 4)

TRANSIENT ISCHEMIC ATTACKS (TIAs)

Management of TIA

بہ ۱۰ سالہ ۱۰ سالہ ہر چھ ماہ بعد ہر چھ ماہ بعد ہر چھ ماہ بعد

DEFINITION

"Mini - Stroke"

"Sudden attacks of temporary cerebral ischemia too short to cause infarction"

- DURATION: few minutes or hours (maximum 24 hours) followed by **complete recovery**.
- VALUE: A TIA is a **warning** of impending ischemic stroke.

- If the attack lasts over 24 hours & then the patient recovers, it is called: **RIND: "Reversible Ischemic Neurological Deficit"**

ETIOLOGY

1. Most commonly:

cerebral emboli arising from:

- Ulcerated atherosclerotic plaques: in the carotid or vertebral arteries.
- Mural thrombi: in a diseased heart.

2. Less commonly:

- Vasculitis** (e.g. SLE & PAN), & **Hyperviscosity** (e.g. Polycythemia).

CLINICAL PICTURE

1. CAROTID TIAs.

(see later)

2. VERTEBRO-BASILAR TIAs.

(see later)

eg - cerebellar ataxia
eg - syncope

اشر جھٹکا
کاملین سے درس
Vascular supply

PROGNOSIS

MCQ

30% of TIA → stroke

- Patients suffering from TIAs are more liable to develop cerebral strokes.

D.D.

1. Other causes of syncope
2. Attacks of hypoglycemia → مرض السكر أخذ حقنة أنسولين غير إكل أو جرعة زائدة
3. Attacks of Epilepsy
4. Other causes of Transient hemiplegia eg Todd's paralysis 53

INVESTIGATIONS

I. BRAIN IMAGING

- ★ 1. Cerebral angiography. - invasive / رخيص
2. CT scan:

to rule out intracranial hemorrhage.

لأنه كل دقيقة تتحرك نحو stroke و حسب huge

II. OTHER IMAGING

1. CARDIAC IMAGING:

Echocardiography (TTE & TEE).

2. VASCULAR IMAGING:

- DOPPLER ultrasonic imaging.

- ★ • MRA / cerebral angiography

radioactive gadolinium صبغة مشعة

MR ولازم صبغة مشعة

non-invasive

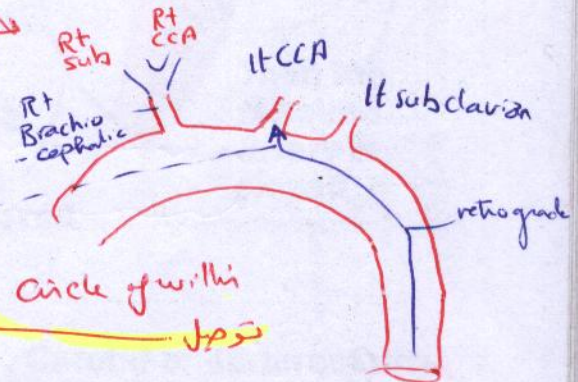
III. LABORATORY TESTS

1. CBC. eg PRV
2. PT & APTT.
3. Risk factors:

e.g. blood glucose, lipid profile, uric acid, homocysteine.

Rt & Lt Carotids
Carotids in V.B.

Circle of Willis
توصيل



TREATMENT

A) Medical:

1. Antiplatelets:

- Aspirin (low dose):
- Dipyridamole (Persantin):
- Ticlopidine (Ticlid):

75 - 300 mg / day (single dose).

75 mg twice daily.

250 mg twice daily.

2. Anticoagulants:

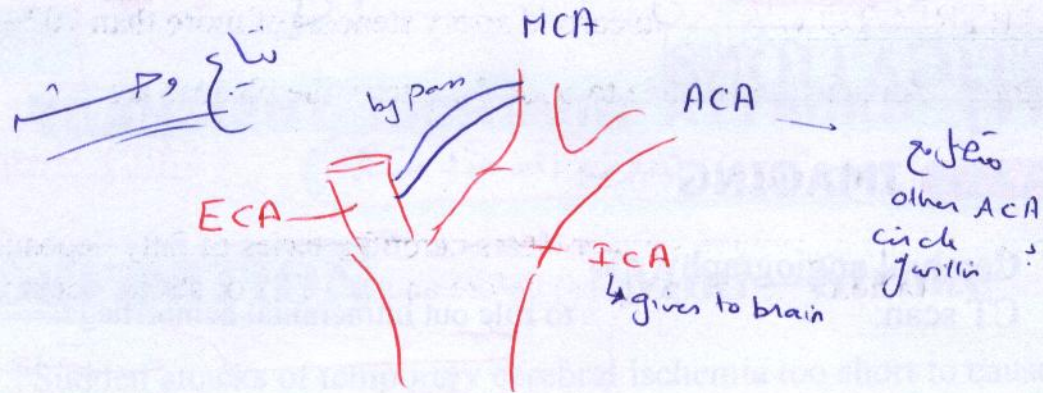
e.g. Dabigatran.

3. Other drugs:

e.g. Trental or Trivastal.

4. Treatment of risk factors.

e.g. proper control of HTN.

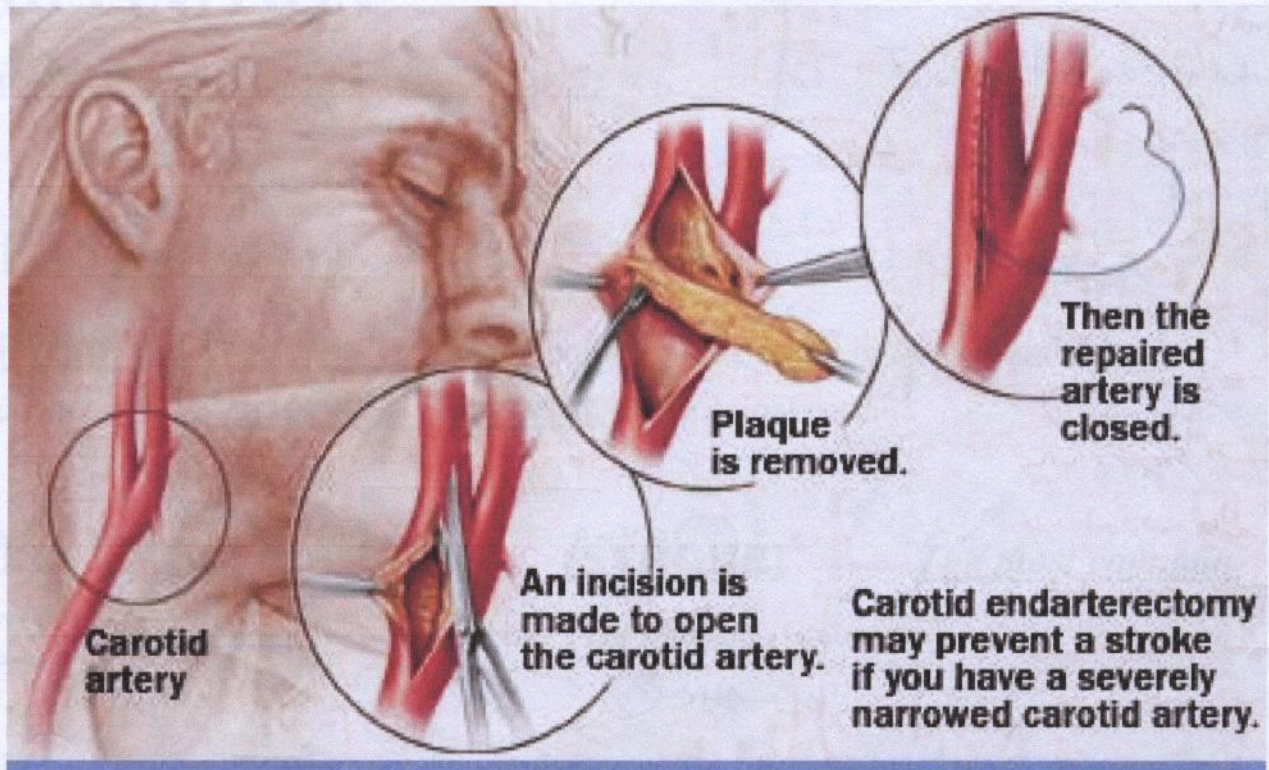


B) Surgical:

1. ENDARTRECTOMY: in carotid artery stenosis of more than 70 %.

Technique: An incision is made to open the artery, the plaques are removed, and the artery is closed.

Value: This preventive surgery clears carotid arteries of fatty deposits (atherosclerotic plaques) before another TIA or stroke occurs.



2. Surgical BYPASS:

- Bypass between the ECA & MCA is generally not beneficial.

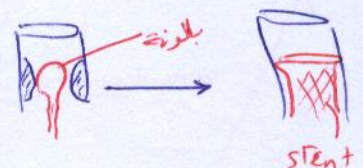
C) Carotid angioplasty & Stent:

Technique:

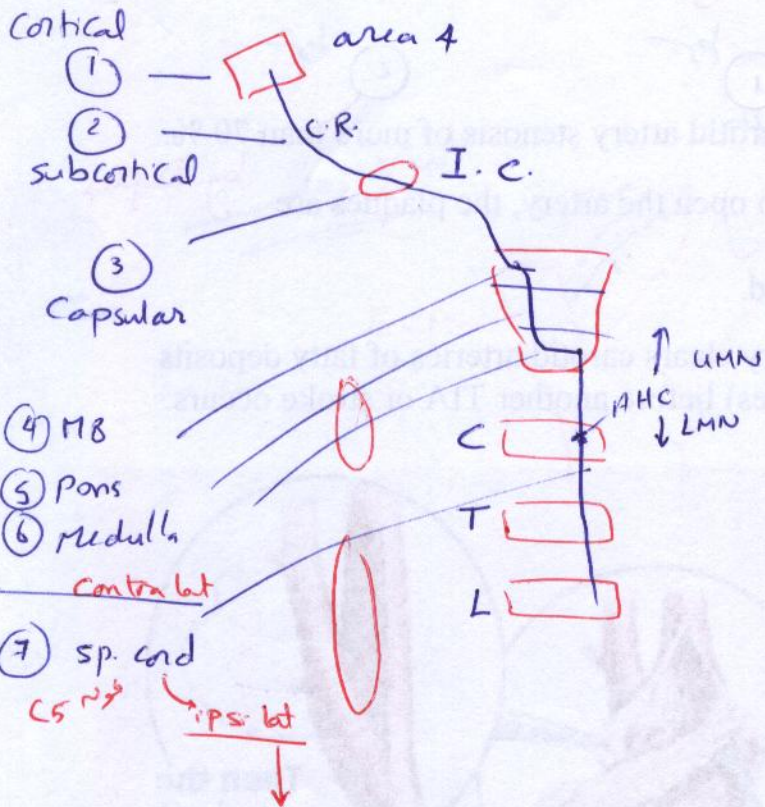
- Angioplasty: Introduction of a balloon to dilate the stenotic artery.
- Stent: Introduction of an intra-luminal Stent to maintain patency of the dilated artery.

Value:

- It maintains patency in the carotid circulation.
- It may offer benefits over surgical procedures.



Cardio و
CABG
Stent
Stent
دلوقة بقى



hemi : نصف

para : 2.L.L.

Quadr: 4 limbs

Mono : 1 limb

* hemiplegia =

UMNL أحلف أو

لا أن ينفصل يكو فيه
Lesion

AHCs
or nerves

of arm & leg
18 segments!

يسمى هذا

غالباً يضر الـ I.C.

لأنه sp. cord

أفلام الماركو

حاجه قدر تشن

على ناحية واحدة

لو نادراً

Brown sequard

والـ AHC كانت متعوده على impulse من LMN

نظراً لضعف

نظراً لضعف

نظراً لضعف

نظراً لضعف

نظراً لضعف

نظراً لضعف

نظراً لضعف

نظراً لضعف

نظراً لضعف

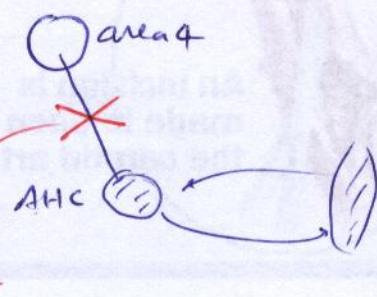
نظراً لضعف

نظراً لضعف

نظراً لضعف

نظراً لضعف

General clip



ضرب LMN
عجالة

* for clinical

grades of Mspower

• Zero : Complete paralysis
"no movement"
موت فني بحركه الهم

• 5 : Normal
= active movement against
gravity & Resistance
(Full Power)

• 4 : = active movement against
G & R
but weaker

• 3 = active movement
against G
not "X"

• 2 = active movement
e gravity eliminated
بحركه الهم

• 1 : flicker of contract
بحركه الهم

White Knight Love

نظراً لضعف

نظراً لضعف

(أما لو ضرب LMN بالشرج من فتوق غير العقل)

then returns → ↑ tone
as no LMN inhibition on it

5: normal
1-4: pariem
Zero: paralysis

Neurone shock
= Transient cessation of function

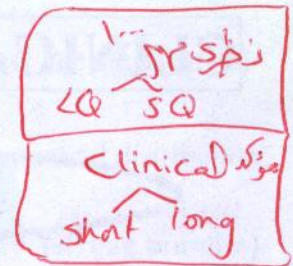
HEMIPLEGIA

DEFINITION

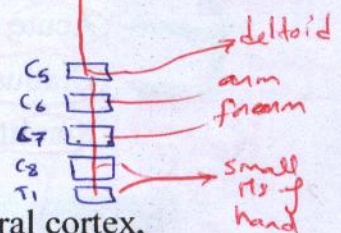
Paralysis of one side of the body. due to unilat Δ tract lesion

ETIOLOGY

Pyramidal tract lesion at any point from its origin in the cerebral cortex, down to the 5th cervical segment of the spinal cord.



لو ضربت تحت C5 ← من كل الذراع هينشل
spared Δ Δ النقيير
deltoid



لو ضربت فوق C5 ← من كل الذراع هينشل
→ Quadriplegia

I. VASCULAR:

(STROKE) ^{etio diagnosis} The most common

- Causes of Stroke: Ischemic (Thrombosis, Embolism), Hemorrhagic.

II. INFECTIVE:

Encephalitis, Meningitis, Brain abscess.

III. NEOPLASTIC:

Glioma, Meningioma.

IV. DEMYELINATING:

= Multiple sclerosis (MS)
Disseminated Sclerosis (DS).
→ characterized by remissions & exacerbations

V. TRAUMATIC:

Cerebral laceration, Subdural haematoma.

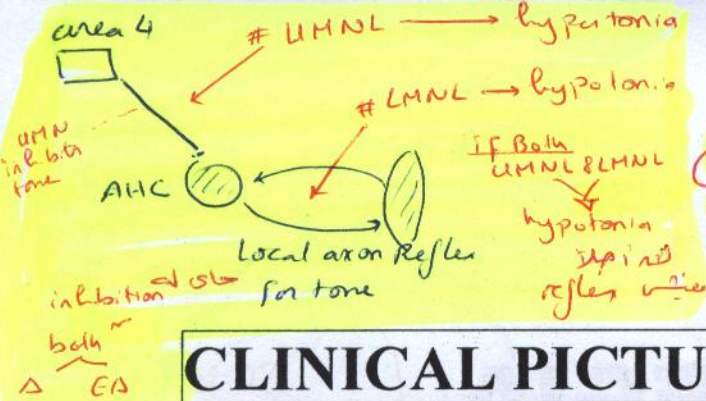
VI. CONGENITAL:

Cerebral palsy.

VII. HYSTERICAL:

Absence of organic pyramidal lesion.

Adherexclerosis لا تشكر أية في Adherexclerosis
SLE فكر صليبي في SLE
White Knight Love
Ethel



صورة عامة للحالة
Cause من الـ level
level

thrombosis
acc to Embolism type
- Cause
- Site = level
7 levels

CLINICAL PICTURE "GENERAL & SPECIFIC"

1. GENERAL CLINICAL PICTURE

Onset & Course

"Vary according to the lesion"

- **Acute** onset & variable course: Vascular, Infective, Traumatic lesions.
- **Gradual** onset & progressive course: Neoplastic lesions.
- **Remittent & Relapsing** course: Demyelinating lesions (DS).

Symptoms & Signs

"Vary according to the onset"

- 1) **Acute lesions:** the hemiplegia passes through 2 stages:
 - I. Stage of **flaccidity**: this is due to neuronal shock.
 - II. Stage of **spasticity**: this is the stage of established hemiplegia.
- 2) **Gradual lesions:** the hemiplegia passes directly to the stage of **spasticity**.

I. Stage of Flaccid Paralysis: (Shock Stage)

neuronal shock

عند حدوث

1. **DURATION:** 2 – 6 weeks, the shorter the duration the better the prognosis.

لوائح من spastic بمرى يقى احسن والعضلات هتبقى مش ضعيفة اوى

2. **On the paralysed side there are:**

- **Muscle tone:** Completely lost. (flaccidity)
- **Deep reflexes:** Absent.
- **Plantar reflex:** Absent. (no Babinski sign)

3. **If the onset is associated with coma,** the paralysed side is determined by:

- The **limbs** on the paralysed side are flaccid & drop passively.
- The **cheek** on the paralysed side moves in & out with respiration.
- There is **conjugate deviation** of the **eyes** to same side of lesion.

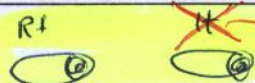
Lateralizing signs

احوار فى السفوى

4. **Finally, RECOVERY** from the shock stage occurs:

- **Muscle tone:** ↑ Reappearance, then gradual increase.
- **Deep reflexes:** ↑ Reappearance, then gradual increase.
- **Plantar reflex:** ✓ Positive Babinski sign.

This means that the Stage of spasticity starts



area 4 → Rt hemiplegia
area 8 → no more gages to Rt so moves to left
lesion

علامات على انها من واحدة
قيمتي ترتك
الناحية بئانه
lesion
له لو الحياه ما من حاله
واختبره لك لو قوما
أستقر فى السفوى

خوار clinical على حالة واحد مسكول يده ررجله

1. clinical diag.: hemiplegia
2. Etio " : Mostly — stroke
3. Anatomical " : site = level (7)
4. Stage " : Flaccid spastic

hemiplegia وتسكت
?? which vessel
?? how affected? → thrombosis, embolism, hyge
التقرن ببول
Blood supply

clonus Babinski

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II. Stage of Spastic Paralysis: (Stage of established hemiplegia)

1. Paralysis of one side of the body: "Paralysis shows a pyramidal distribution"

الاعرج الجاذبية ضيف املا
- Affects the **progravity** (weak muscles) more than the **antigravity** (strong muscles).

In UL: the **extensors** (progravity) are weaker than the flexors

In LL: the **flexors** (progravity) are weaker than the extensors

UL F → anti
E → pro
LL F → pro
E → anti (Quadriceps)

- Affects the **distal** more than the **proximal** muscles.

Oral diseases affect

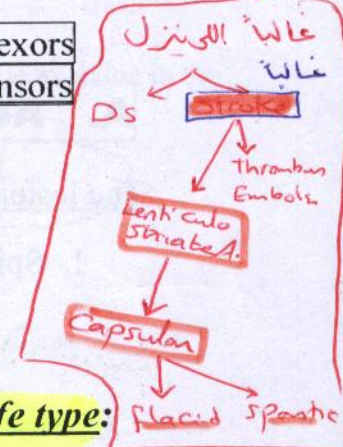
Distal Pyramidal PN - except →

Proximal Myopathy Myasthenia GBS

The **hand** is weaker than the shoulder

The **foot** is weaker than the hip.....

if acute → flaccid → spastic
if gradual → spastic



2. Hypertonia (spasticity) of the paralysed side of clasp-knife type:

- Affects **antigravity** (stronger muscle tone) more than **progravity** (weaker muscle tone).

In UL: the **flexors** (antigravity) are more spastic than the extensors

In LL: the **extensors** (antigravity) are more spastic than the flexors

الاعرج (anti) أقوى

الاعرج حاجة وانت بتقل extension

الاعرج حاجة وانت بتقل flexion

3. Exaggerated deep reflexes:

- Deep reflexes in both UL & LL are **exaggerated** on the paralysed side.
- Pathological deep reflexes (normally absent) may appear.
- Clonus may be present.

وضع رجلاه ثاني يده وفارده رجلاه

oral & clinical

- انواع hypertonia
1. clasp knife spasticity (Δ)
 2. lead pipe or cogwheel rigidity (EΔ)
 3. Decerebrate rigidity
 4. Decorticate "

4. Lost superficial reflexes.

5. Extensor Plantar reflex. (Positive Babinski sign)

6. Gait: (Circumduction)

If the patient can walk, his gait is **circumduction** due to spasticity of the **extensors** of LL.

Paresis not paralysis

If Bilat. Paraplegia

طرح دي طرح دي

57

Scissor White Knight Love

منعرجات
يستعمل (flexors)
فينظم حركه كل رجلاه كتلة واحدة ويصير ليده

3 at cerebral $\Rightarrow$ Before cross
 3 at B-stem $\Rightarrow$ After cross
 1 at sp. cord $\Rightarrow$ After cross

نظري 2
 1- 2
 3- 4

58

2. SPECIFIC CLINICAL PICTURE

It varies according to:

- A. SITE OF LESION. B. CAUSE OF LESION.

A. ACCORDING TO THE SITE OF THE LESION

The lesion causing hemiplegia may occur at 3 main levels: "LEVELING"

- I. Spinal cord. II. Brain stem. III. Cerebral.

I. SPINAL HEMIPLEGIA (Brown-Sequard Syndrome)

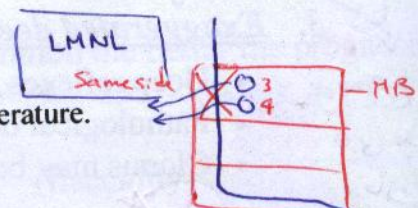
- The lesion is on one side of the cord & is situated between C1 & C5 segments.
- It is caused by: stab wound, disc prolapse, DS or tumour.

a) At the Level of the Lesion:

- Ipsilateral localised LMNL of the muscles supplied by the affected segments.
- Ipsilateral loss of all sensations in the area supplied by the posterior roots of the affected segments.

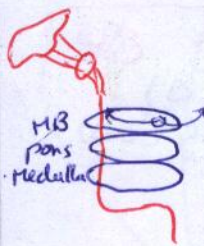
b) Below the Level of the Lesion:

- Ipsilateral hemiplegia (UMNL).
- Ipsilateral deep sensory loss.
- Contralateral superficial sensory loss for pain & temperature.
- Touch diminishes on both sides.



II. BRAIN STEM HEMIPLEGIA (Crossed Hemiplegia)

- Hemiplegia on the OPPOSITE SIDE of the lesion.
- Cranial nerve paralysis of LMN nature on the SAME SIDE of the lesion.



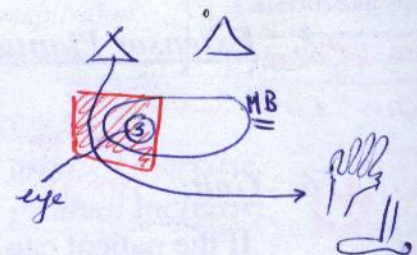
1. Mid-Brain Lesion

a) Weber's syndrome:

- Hemiplegia on the opposite side of the lesion.
- 3rd cranial nerve paralysis on same side of lesion.

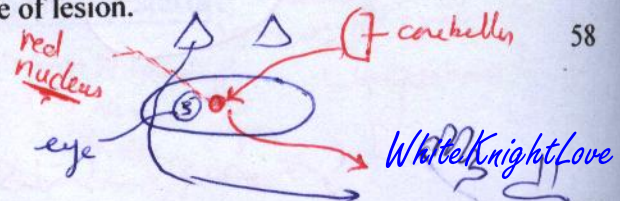
b) Benedict's syndrome:

- Hemiplegia & Hemiataxia on the opposite side of the lesion.
- 3rd cranial nerve paralysis on the same side of lesion.



حرك العين
 في الاتجاه
 3- 4
 5- 6

هذا لم يضر cerebellum
 Cerebellum fibers $\rightarrow$ الى رايه اي
 Red nucleus $\rightarrow$ Red nucleus
 cross $\rightarrow$ cross

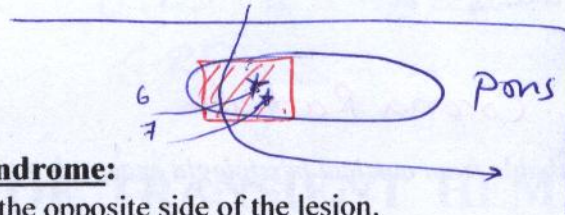


58

• conjugate move of eyes
by area 8 + MLB

59

2. Pontine Lesion

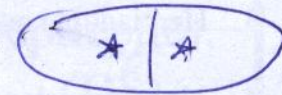


a) Millard Gubler Syndrome:

1. Hemiplegia on the opposite side of the lesion.
2. 6th & 7th cranial nerve paralysis on the same side of lesion.

b) Foville Syndrome:

1. Hemiplegia on the opposite side of the lesion.
2. Loss of conjugate deviation of the eyes to the same side of the lesion due to lesion in the medial longitudinal bundle (MLB).



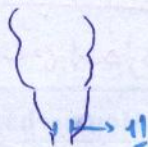
3. Medullary Lesion

a) Avellis Syndrome:

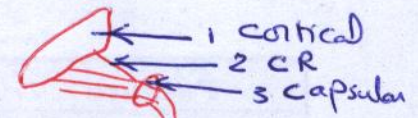
1. Hemiplegia on the opposite side of the lesion.
2. 9th & 10th cranial nerve paralysis on the same side of the lesion.

b) Jackson's Syndrome

1. Hemiplegia on the opposite side of the lesion.
2. 10th & 12th cranial nerve paralysis on the same side of the lesion.



مغزى !! لائن بعد موز

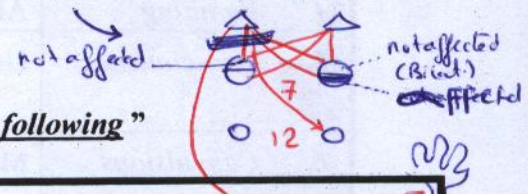


III. CEREBRAL HEMIPLEGIA

① opp. Lesion in the cerebral hemisphere results in hemiplegia (associated with UMN facial and hypoglossal paralysis) on the opposite side of the lesion, but without any cranial nerve paralysis on the same side of the lesion.

③ opp

② opp



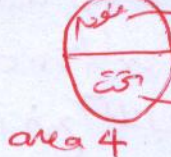
1. Cortical:

"characterised by one or more of the following"

1. Coma if the lesion is extensive. *غيبوبة*
2. Convulsions if the lesion is irritative. *نوبات صرعية*
4. Contralateral cortical sensory loss: if the lesion involves the parietal lobe. *الحس في النصف المقابل*
4. Homonymous hemianopia: *LIR* if the lesion involves the occipital lobe. *Macula* *area 17*
5. Aphasia and agraphia: *(44, 45 on lateral)* if the lesion is in the dominant hemisphere. *44, 45*
6. The paralysis usually involves one limb (monoplegia) specially in vascular lesions. *كتابة كلام*

(incomplete hemiplegia)
ذراع يسار او رجل يسار
هناك بعض حالات لايم
hemiplegia

التعب



يد
رجل

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* بتر ربنا انه الـ A. يتابع لعض الفوقاني
غير A. يتابع لعض التحتاني
الـ lesion يربط بين الطرفين الثاني:
hematoma extends in White Knight Love
or thrombus extends in Arley hill foot

فبرس
لأنه على تخریب فی
لکونه کبره اودی
وتعمل السطح المائل فی الحیم
ولو هو بجحر حریة
area 4 متفاحه فی

60

2. Subcortical:

Corona Radiata

- It is indistinguishable from cortical hemiplegia except that the paralysis is more extensive.

3. Capsular:

"characterised by the following"

1. Complete hemiplegia associated with UMN facial and hypoglossal paralysis:
"All are on the opposite side of the lesion".
2. Hemihyposthesia on the opposite side of the lesion.
3. Hemianopia may occur, if the fibres of the optic radiation in the capsule are involved.

4. No convulsions,

No aphasia,

No coma.

irritative lesion
Convulsions
irritate فی

44
موجوده فی
cortex

I.C. لاه
مالوش دعوه
بال cortical
neurons
RAS لا فی

B. ACCORDING TO THE CAUSE OF THE LESION

- As the vascular causes are the commonest causes in hemiplegia, it is important to differentiate the clinical picture in thrombotic, embolic and hemorrhagic lesions.

لازم یکتب فی انجری

Feature	Thrombosis	Embolism	Hemorrhage
1. Age	Old age	Any age	Commonly old age
2. Onset	Rapid (taking hours)	Sudden (taking sec.)	Dramatic or apopleptic
3. Prodromata	TIA's	Absent TIA's ✓	Absent
4. Vomiting	Absent	Absent	Common pressure on center
5. Consciousness	Usually preserved	Usually preserved	Lost with deepening coma compress cortical neurons
6. Convulsions	May occur	May occur	Frequent mass e irritative effect
7. Pupils	Normal and equal	Normal and equal	Dilated and irreactive
8. Fever	Absent	Absent	Present irritates HRC in hypothalamus
9. Blood pressure	May be high	Normal	Usually high
10. Heart	May be cardiac insufficiency	Usually valvular lesion / mural thrombus / Af	Left ventricular hypertrophy
11. CSF	Clear	Clear	Bloody, ↑ tension
12. CT scan, MRI	Hypodense area	Hypodense area	Hyperdense area

obscure

invasive
وعی
CT & MRI

الکلام فقط فی CT و MRI (افس)

Hge may leak in ventricles

White Knight Love
افض الیایه 4.5 mm
سپرد
حتی کرم انشی

60

[① . DD } TIA,
 [② . oral & clinical
 [③ . GRF }

61

CAUSES OF TRANSIENT HEMIPLEGIA

1. Transient ischemic attacks (TIAs).
2. Todd's paralysis (post-epileptic).
3. Demyelinating disease (DS).
4. Hemiplegic migraine.
5. Hysterical.

< 24 hr

$\frac{1}{4}$ - $\frac{1}{2}$ hr.

< 24 hrs
N. G.!

neurons
or
jacksonian
fit.

relapse & remission

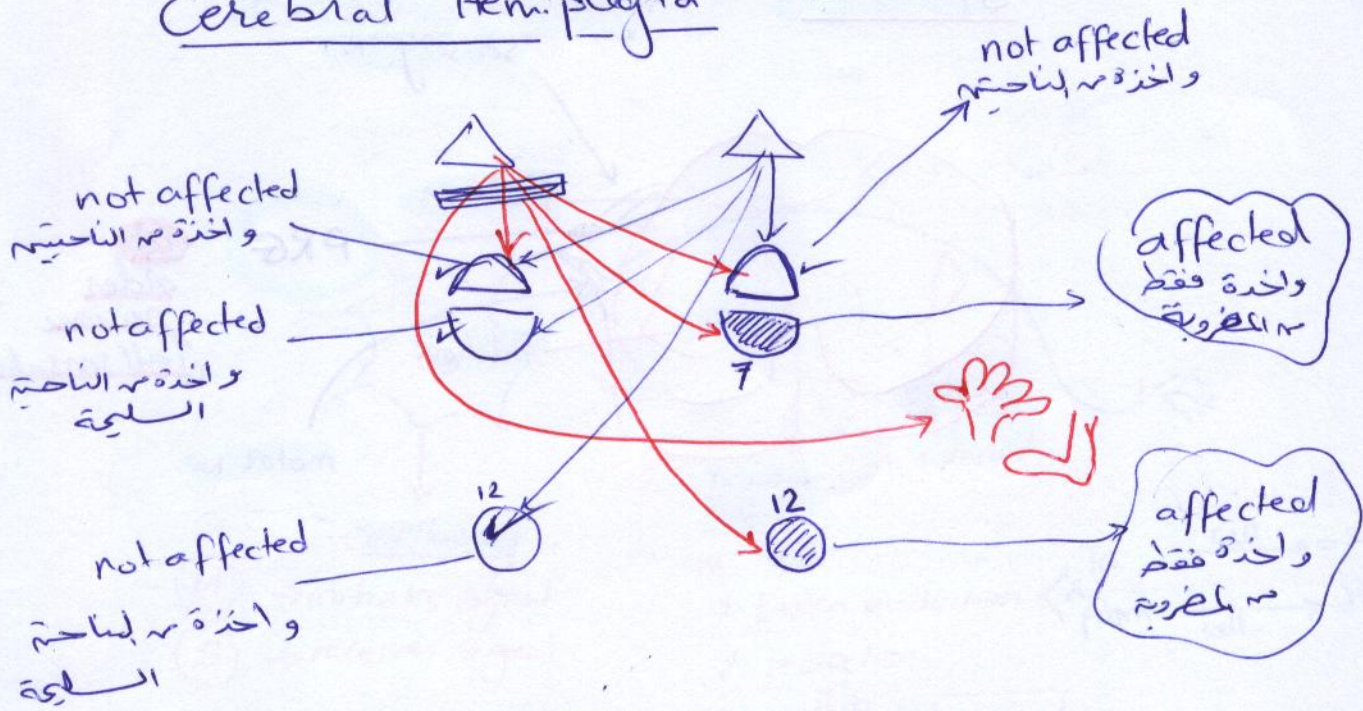
لما تزعل بس

من محقول
نقص
فيتيل
نادر أحيث

transient hemiparesis

Cerebral Hemiplegia

دس حور عاة
for cerebral



Special sense VIII صوت
II ذوق

Coma in Cortical

normally

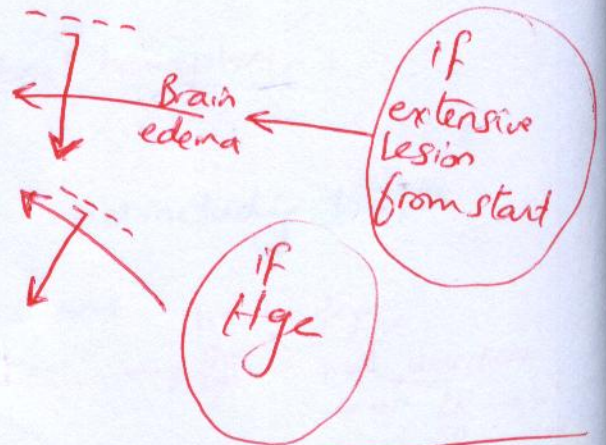
RAS

إشارة

Cerebral cortex
"cortical neurons"
فتقى

التقىير

extensive lesion
if **logic**



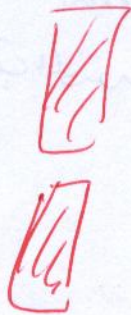
aphasia

Q - من غير أن يكون hemiplegia إلى جانب aphasia

A - الإجابة: هو الحبل
 إلى عنده hemiplegia
 في يده ورجله
 إلى يتحكم
 (Rt in most people)
 hand leg

Rt

Lt
 44



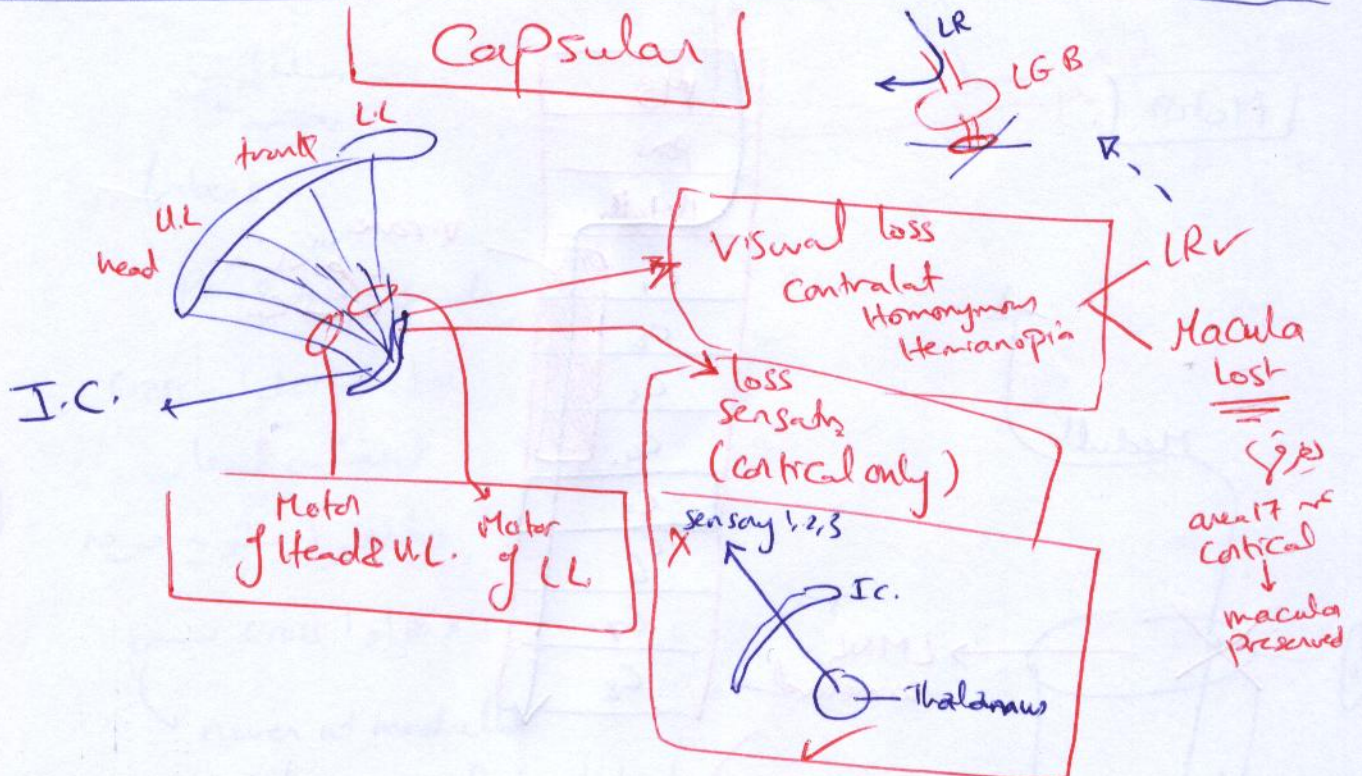
Aphasia

Oral

أما لو أجزء عنه في Rt
 not dominant

فمنه لرجله و يده. الحبل و عنده Aphasia

Capsular



Cortical

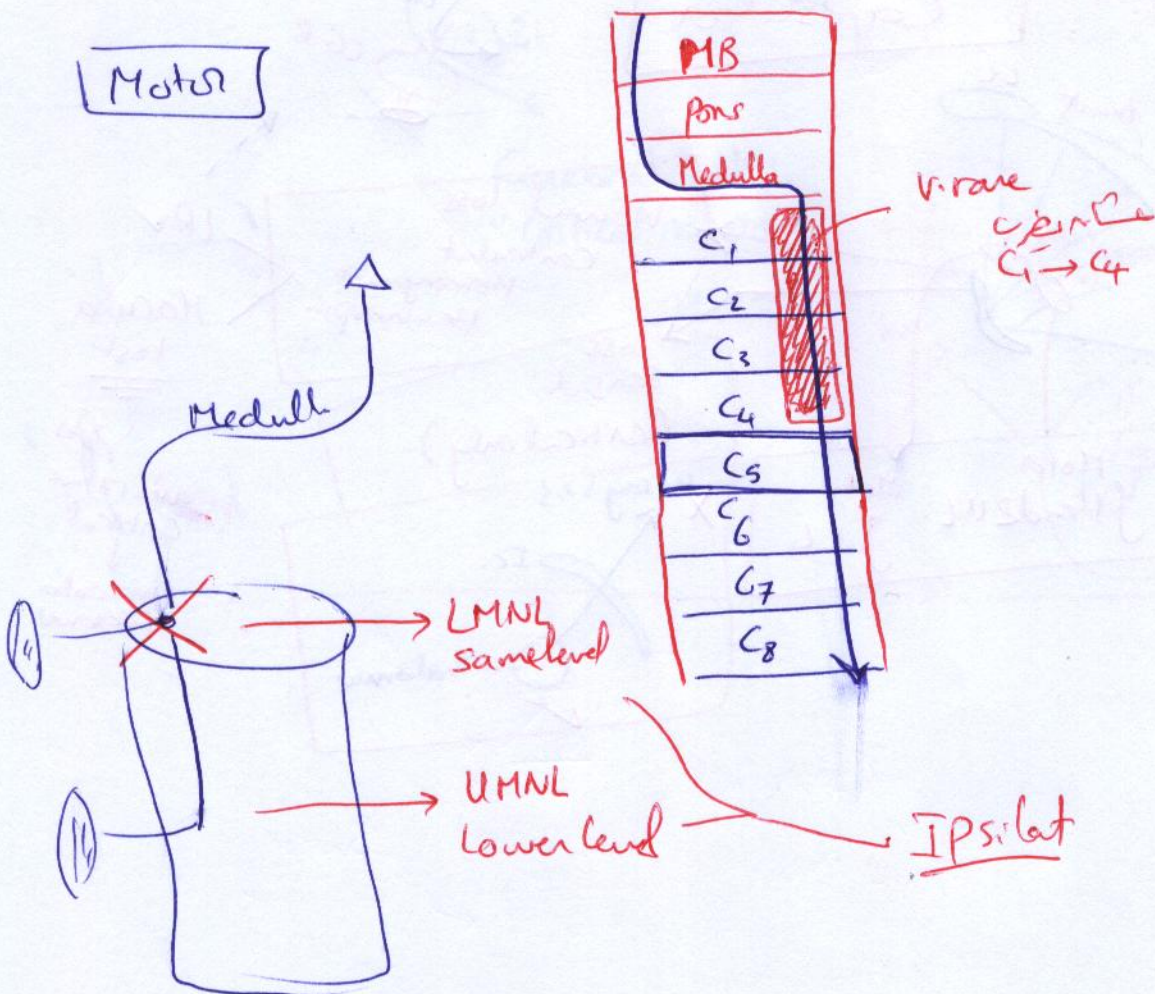
- Monoplegia
- Maybe
Coma
Convulsions
Aphasia
- Macula preserved

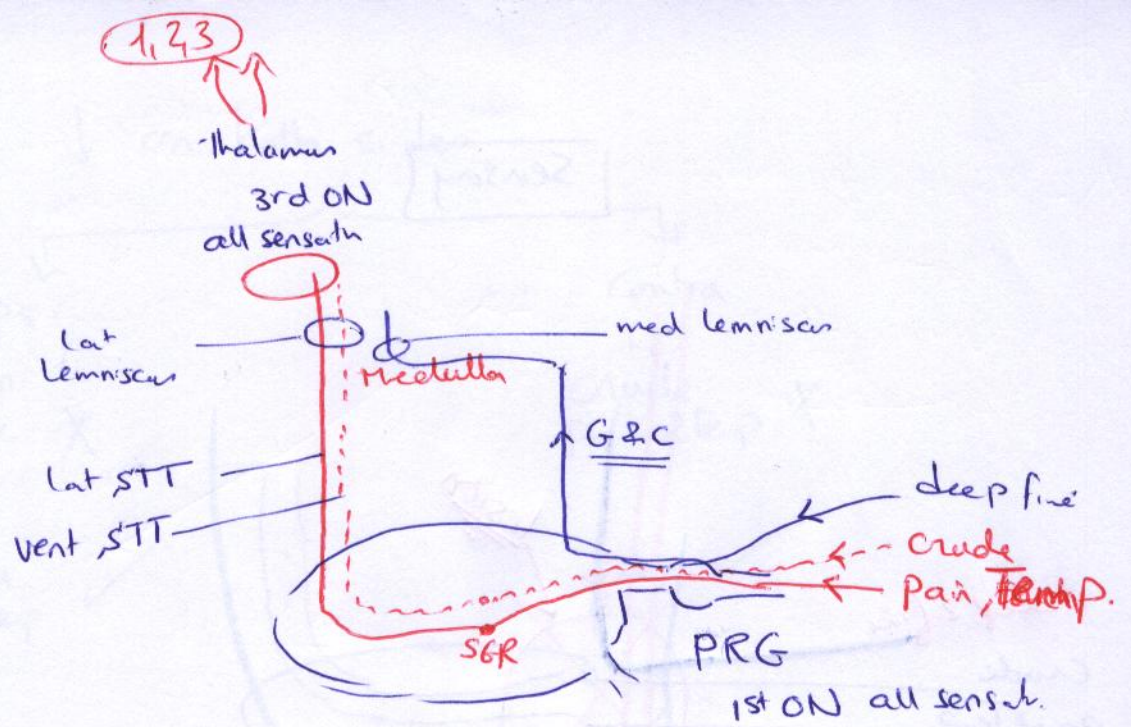
Capsular

- Complete hemi
ip^s of right & left
- Never
Coma
Convulsions
Aphasia
- Lost

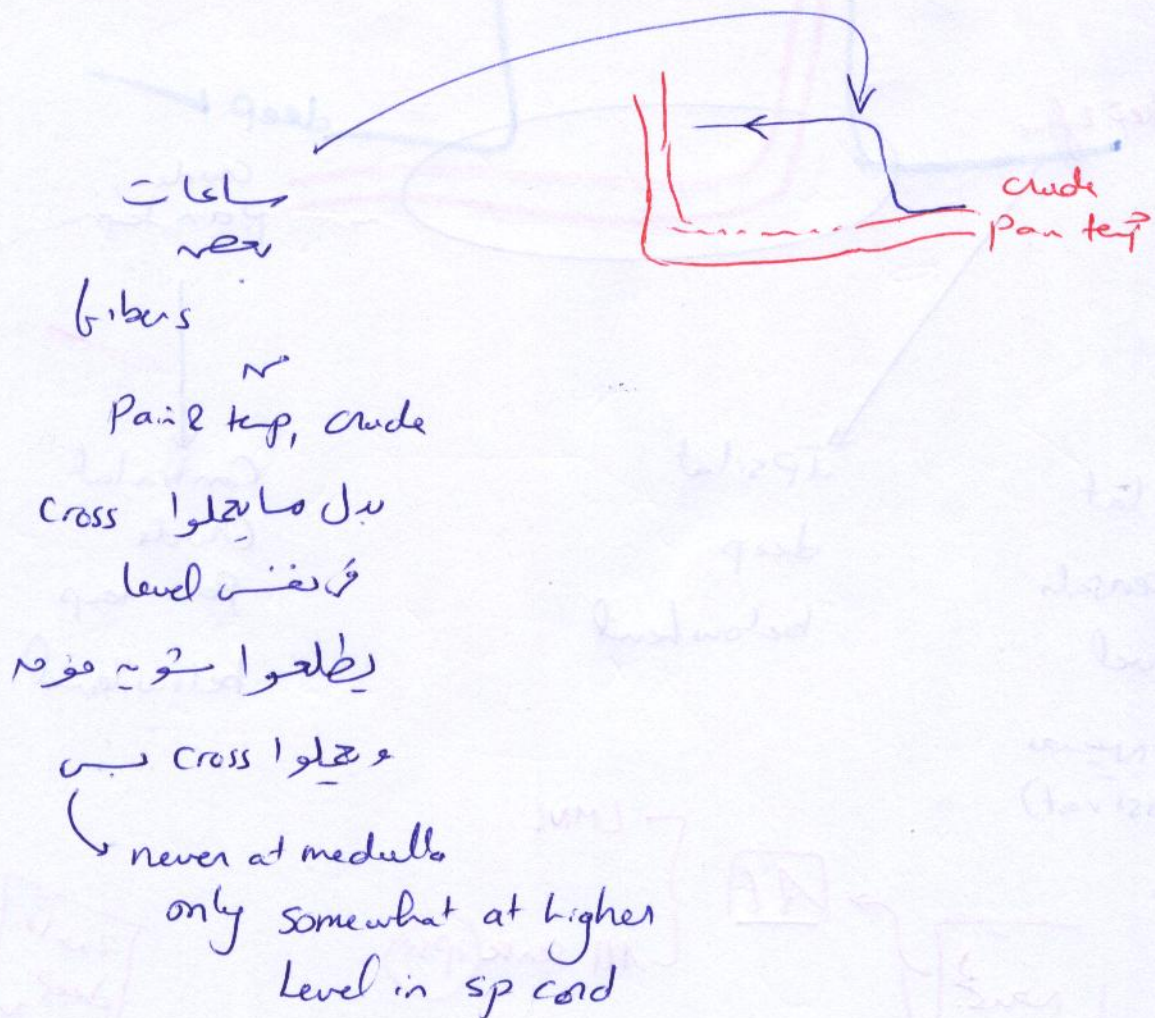
Spinal hemiplegia

Motor

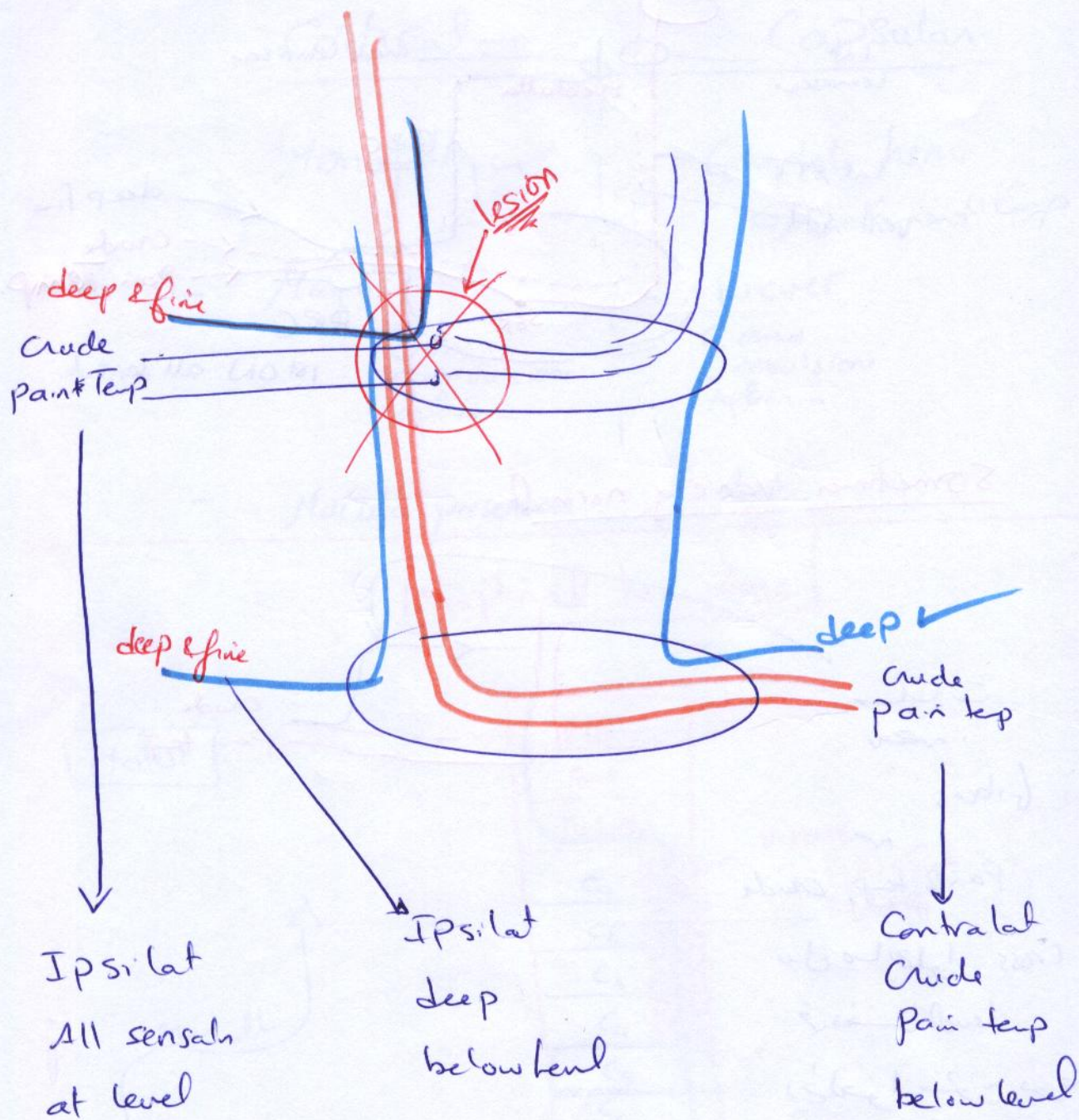




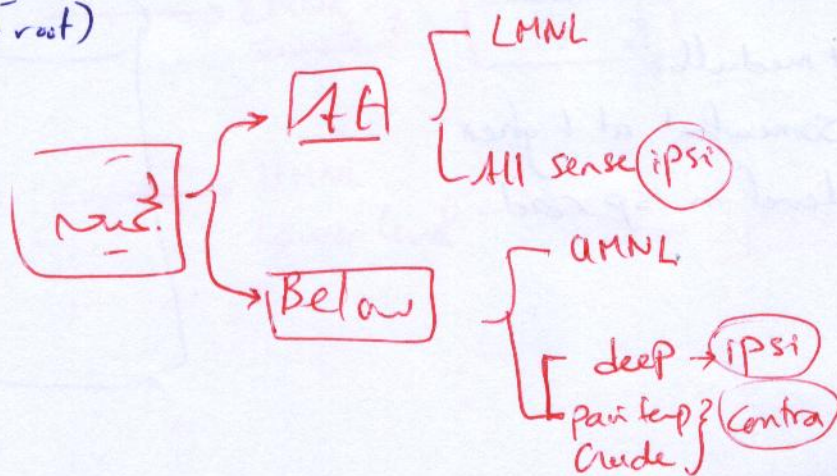
Sometimes variety normal



Sensory

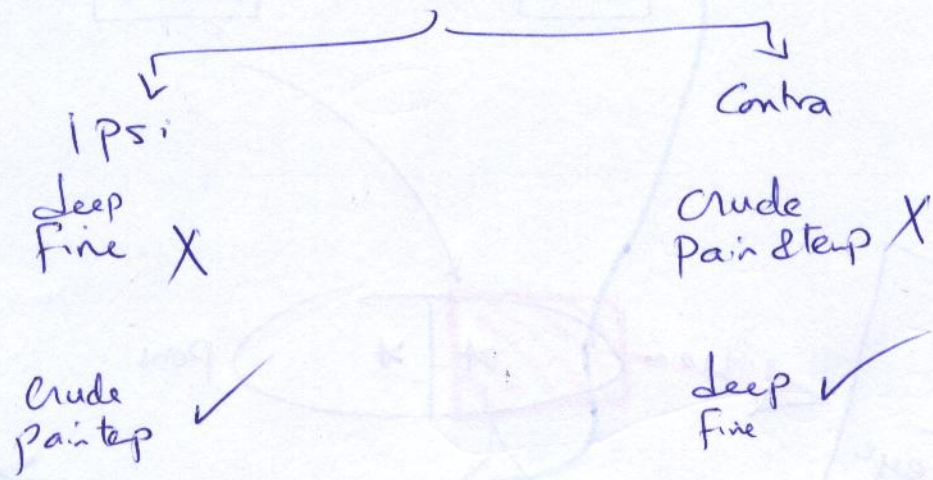


is Neuro Post root)



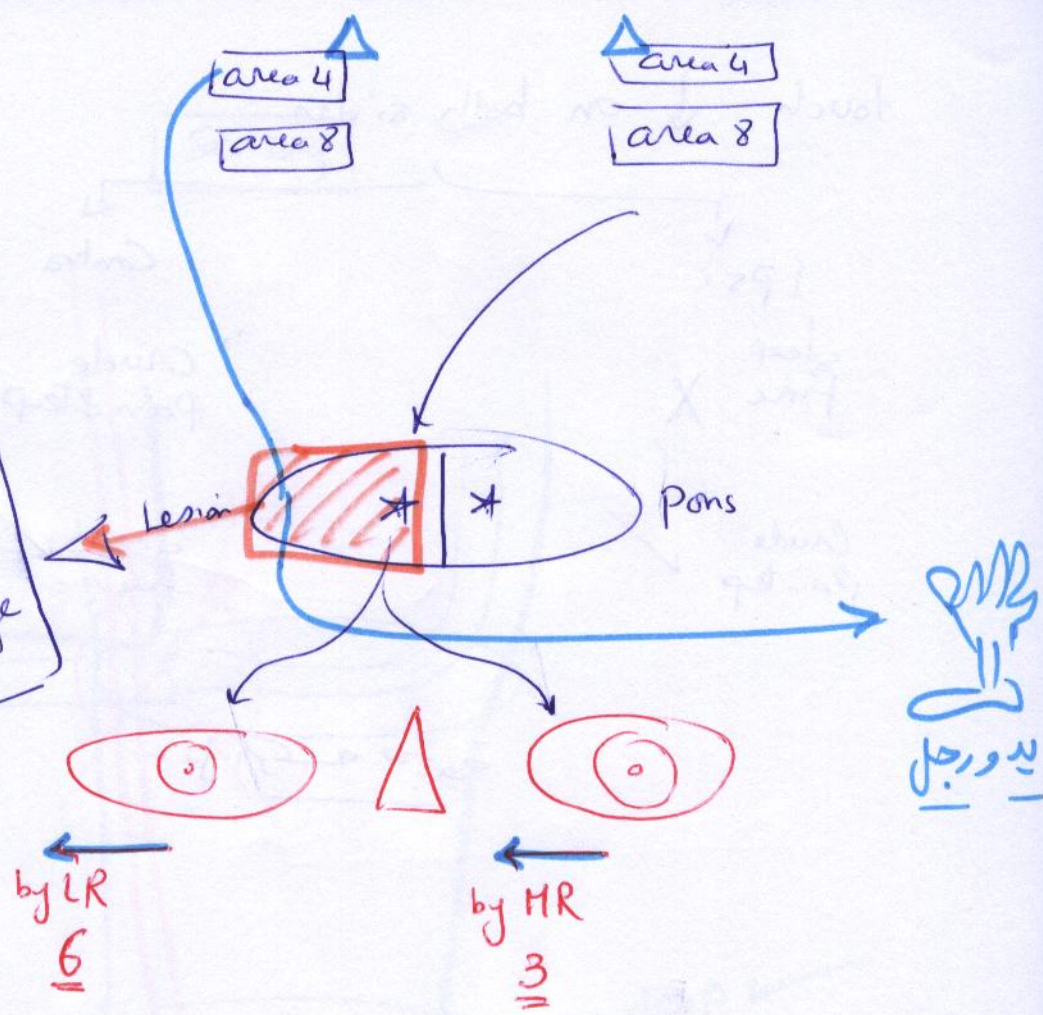
3 to deep ~

touch ↓ on both sides



پہلے سے پہلے

hemiplegia
on opposite
no conjugate eye
to same side



Q Discuss ICA GCR

1 Anatomy

2 C/P

TIA
stroke

discuss details of hemiplegia

62

BLOOD SUPPLY OF THE BRAIN

① General C/P (placed & spastic)

② specific C/P

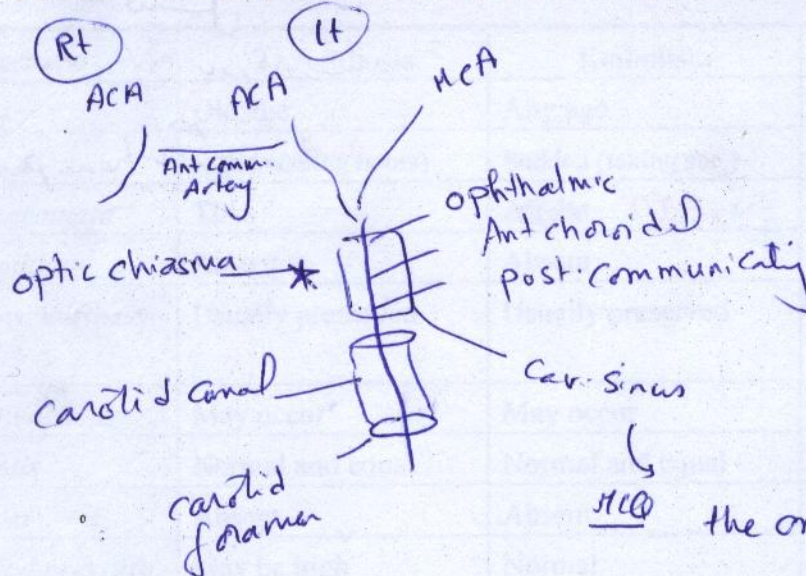
The blood reaches the brain through two Systems of blood vessels:

spinal لاشرح
by V.B. ← Brain stem لاشرح
Level : cerebral only ← لاشرح
Cause

I. THE CAROTID SYSTEM.

Thrombosis
Embolism
hge.
لو حاتم الوال
occlusion
لو حاتم
الوال
lesion

II. THE VERTEBROBASILAR SYSTEM.



the only anat. locatn

At w A. passes through
venous Blood

لو A. فرج والبال عات
A-v. fistula

2 Communicating:

Ant. → joins 2 ACA
Post → joins Carotid & V.B.

63

I. THE CAROTID SYSTEM

INTERNAL CAROTID ARTERY

ANATOMY

Each internal carotid artery enters the cranial cavity through the: Carotid foramen and Canal to the Cavernous sinus where it lies lateral to the optic Chiasma.

The artery in the sinus gives off 3 SMALL BRANCHES:

- The ophthalmic artery.
- The anterior choroidal artery.
- The posterior communicating artery.

Q: oral
PCA: post comm. A.
V.B. & ICA ~ فرع
التي: الـ

The artery finally divides into its 2 MAIN TERMINAL BRANCHES:

The middle cerebral artery and the anterior cerebral artery.

INTERNAL CAROTID ARTERY

OCCLUSION

- **Etiology:** most commonly atherosclerosis of the artery. 99%
- **Age:** most commonly the 4th-6th decades.
- **Sex:** most commonly affecting the males.

ischemia في
thrombosis في
is on top
of Atherosclerosis

- **Clinical Picture:**

1-1/2 hr
< 24 h
Hypoxia → V.D → stretch meninges

I. Recurrent Carotid TIAs:

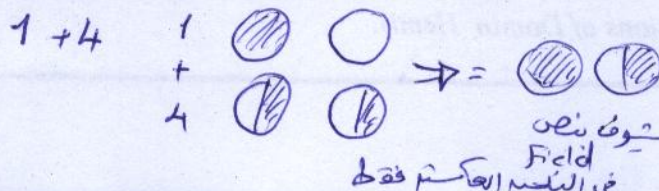
1. Transient sudden Headache.
2. Transient Convulsions. (Transient brain hypoxia)
3. Transient Ipsilateral Blindness. (Transient ischemia of ophthalmic A.)
4. Transient Contralateral Hemiparesis & Hemihyposthesia. (Transient ischemia of sensory pathway of cortical capsule)
5. Transient Aphasia in lesions of the DOMINANT HEMISPHERE.. (Transient ischemia of motor pathway)
6. "These manifestations indicate insufficiency of the Internal Carotid Artery, (Carotid TIAs) which may terminate in complete occlusion of the artery."

"may precede complete occlusion"
warning of impending stroke

II. STROKE (Complete Carotid Occlusion):

1. Ipsilateral Blindness. (> 24 h)
2. Contralateral Hemiplegia & Hemihyposthesia.
3. Aphasia in lesions of the DOMINANT HEMISPHERE.
4. Contralateral Homonymous Hemianopia.
5. INTERNAL CAROTID PULSE: diminished or lost + audible bruit.

Pulse
ضعيف



63

WhiteKnightLove

1. THE MIDDLE CEREBRAL ARTERY ^{eg: 11}

It runs on the lateral surface of the cerebral hemisphere.

It supplies the lateral aspect of the anterior 3/5s of the cerebral of hemisphere.

It gives the following branches:

مناطق علة بال occipital

1. CAPSULAR BRANCH

2. CORTICAL BRANCHES.

1. CAPSULAR BRANCH (Lenticulo-striate artery):

- It supplies all the dorsal half of the IC.

ANATOMY

CAPSULAR BRANCH (Lenticulo-striate artery):

- It is the most commonly occluded artery resulting in:

OCCLUSION

1. Contralateral complete hemiplegia affecting the UL & LL to the same extent.
2. Contralateral UMNL of the facial & hypoglossal nerves.
3. Contralateral hemihyposthesia of the cortical type.
4. Contralateral hemianopia may occur.
5. No coma, No convulsions, No aphasia.

2. CORTICAL BRANCHES:

CORTICAL BRANCHES:

ANATOMY

OCCLUSION

Frontal branch

- Motor area of face & UL
- Motor speech area (44)
- Motor writing area (45)

Frontal branch

Contralateral Faciobrachial monoplegia
Motor aphasia (in lesions of Domin Hemi)
Agraphia (in lesions of Domin Hemi)

Parietal branch

- Sensory area of UL
- Angular gyrus (39)
- Supramarginal gyrus (37)
- Upper fibres of optic radiation

Parietal branch

Cortical sensory loss in UL (but felt in thalamus)
Alexia (in lesions of Domin Hemi)
Apraxia of both sides (in lesions of Domin Hemi)
Contralateral Lower quadrantic Homo Hemianopia

Temporal branch

- Auditory areas
- Lower fibres of optic radiation

Temporal branch

Auditory agnosia (in lesions of Domin Hemi)
Contralateral Upper quadrantic Homo Hemianopia

MAIN ARTERY OCCLUSION

1. Coma & may be Convulsions at the onset (because the lesion is extensive).
2. Contralateral hemiplegia affecting UL more than LL. [capsular: UL + LL, cortical: UL]
3. Contralateral hemihyposthesia with more cortical sensory loss in UL. UL > LL
4. Contralateral homonymous hemianopia.
5. APHASIA (Motor aphasia, Agraphia, Alexia, Auditory agnosia) and APRAXIA (in lesions of Domin Hemi).

2. THE ANTERIOR CEREBRAL ARTERY

It runs on the medial surface of the cerebral hemisphere.

It supplies the medial aspect of the anterior 3/5s of the cerebral hemisphere.

It gives the following branches:

1. CAPSULAR BRANCH

1. CAPSULAR BRANCH (Heubner's artery):

- It supplies the ventral half of the anterior limb of the IC.

ANATOMY

CAPSULAR BRANCH (Heubner's artery):

- Contralateral Facio-brachial monoplegia.
(proximal more than distal)

OCCLUSION

2. CORTICAL BRANCHES:

ANATOMY

Frontal branch

Prefrontal area

- Mentality
- Personality
- Inhibition of primitive reflexes

Paracentral branch

- Motor area of LL
- Sensory area of LL
- Paracentral lobule

Callosal branch

- Corpus callosum

2. CORTICAL BRANCHES.

CORTICAL BRANCHES:

OCCLUSION

Frontal branch

Prefrontal area

Mentality changes
Personality changes
Positive grasp reflex

Paracentral branch

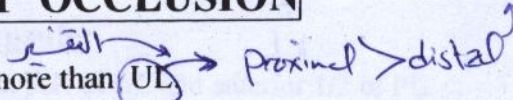
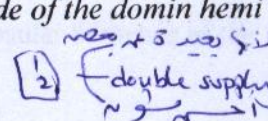
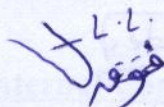
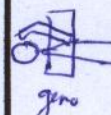
Contralateral Monoplegia in LL
Contralateral Cortical sensory loss in LL
Incontinence of urine (in bilateral lesions)

Callosal branch

Apraxia of the same side of the domin hemi

MAIN ARTERY OCCLUSION

1. Contralateral hemiplegia affecting LL more than UL
2. Contralateral cortical sensory loss in LL.
3. Mentality & Personality changes & Positive grasp reflex.
4. Incontinence of urine (in bilateral lesions).
5. Apraxia of the same side of the domin hemi.



NB

Lesion [1] : All sensory ✓
cortical

Lesion [2] → ^{قشر} sensory
cortical ✓

Lesion [3] : All sensory X

I.C.

Caudate

Lentiform

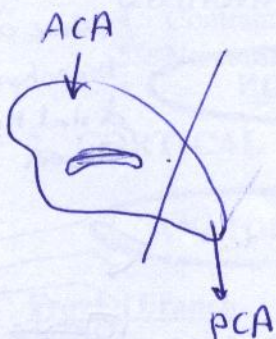
Thalamus

1, 2, 3

All
sensations
lost
• Parietal & Caudate
is no loss
• deep & fine
Medulla is lost

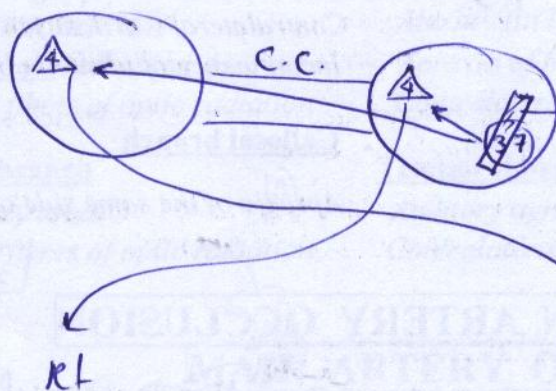
Medial (cortex)

Lateral (cortex)



Comment on MCA cortical bi.:

apraxia on both sides



by MCA lesion

فقدت
motor area 4
(Bilat)

Corpus callosum

Loss of voluntary complex movement on

Same side of dominant hemisphere.

LT

WhiteKnightLove

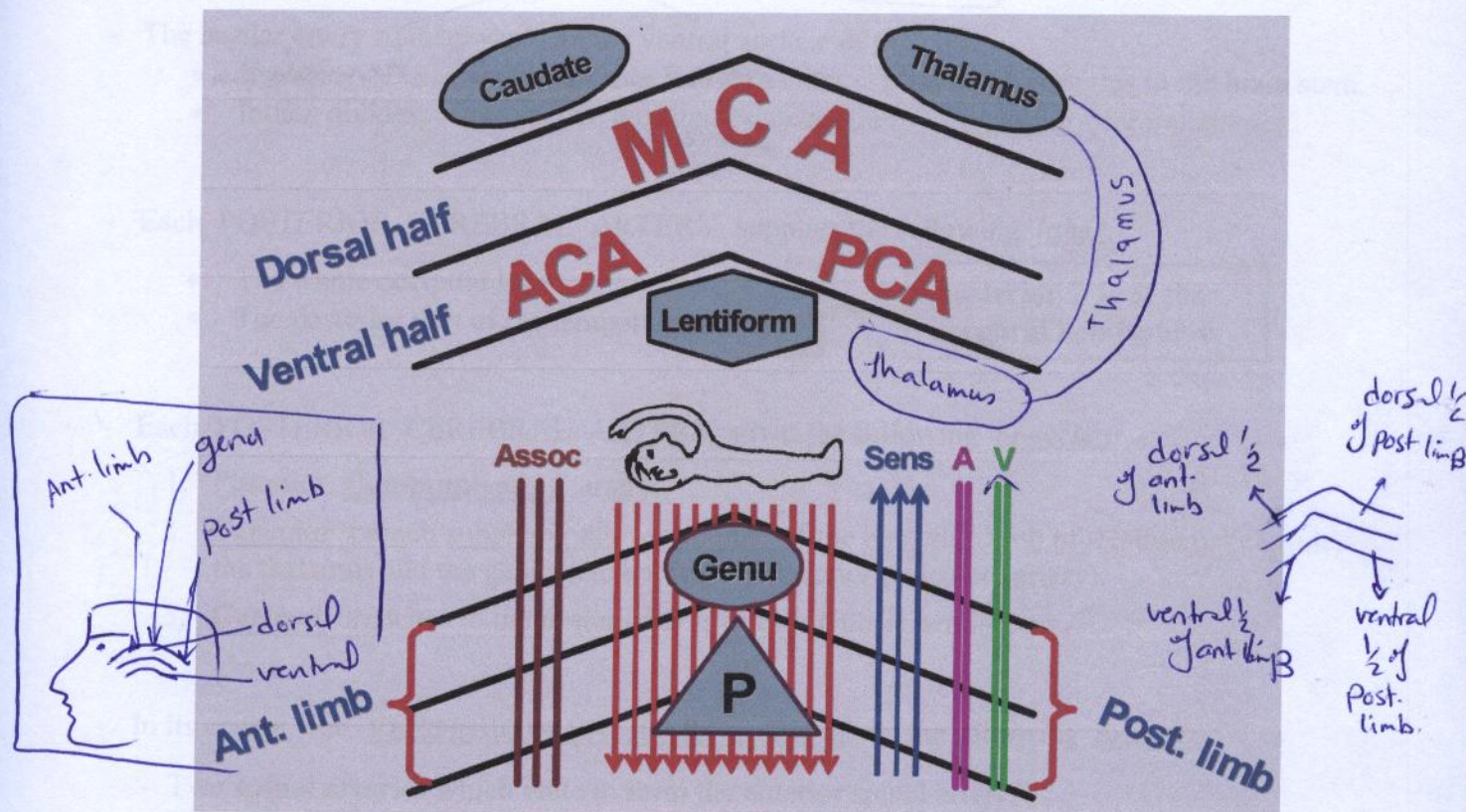
INTERNAL CAPSULE

- It is a broad band of white fibres lying in the depth of the cerebral hemisphere.

It is formed of:

1. The anterior limb: placed between the caudate nucleus and the lentiform nucleus.
2. The genu.
3. The posterior limb: placed between the thalamus and the lentiform nucleus.

ایمان - ناحیه اوتس
Cerebellum ناحیه



Blood Supply of the Internal Capsule:

1. The dorsal half of the anterior limb, knee, and posterior limb of the internal capsule is supplied by the Capsular branch of the middle cerebral artery (**Lenticulo striate artery**).
2. The ventral half of the anterior limb is supplied by the capsular branches of the anterior cerebral artery (**Heubner's artery**).
3. The ventral half of the knee and posterior limb is supplied by the capsular branches of the posterior cerebral artery (**Thalamo-geniculate artery**).

Fibres passing through the Internal Capsule:

1. Pyramidal fibres descend in the genu, adjacent part of AL and anterior 1/2 of PL.
The fibres supplying the arm are followed by those supplying the head, trunk, and lastly the LL.
2. Sensory fibres from the thalamus ascend in the PL.
3. Auditory and optic radiations pass in the posterior part of PL.
4. Associative fibres pass in the anterior part of AL.

lost if either lesion MCA or PCA
from LGB towards occipital lobe

Causes of Paraplegia

- Contra ~~Complete~~ hemiplegia
- Contra

if ^{Ant} spinal A. occlusion → Paraplegia
& another cause

paraplegia $\leftarrow$ loss of all sensation

!ie sensatⁿ of feet,
deep & fine as post column is spared
dissociated sensory loss

Oral if post inf. cerebellar A. occlusion?
(PICA)

II. VERTEBRO-BASILAR SYSTEM

ANATOMY

- Each **VERTEBRAL ARTERY** passes upwards through the vertebral foramina to enter the cranial cavity through the foramen magnum and runs upwards on each side of the medulla.
- Both vertebral arteries meet at the lower border of the pons to form one midline single artery: the **BASILAR ARTERY**. *ponto medullary jn*
- The basilar artery runs upwards on the ventral surface of the pons.
 - It gives: Small branches known as the: *short circumferential* **paramedian arteries** to the **brain stem**.
 - It then divides: into its two terminal branches the: **posterior cerebral arteries**.

- Each **POSTERIOR CEREBRAL ARTERY** supplies the following **lobes**:

- The whole occipital lobe.
- The posterior part of the temporal lobe.

posterior 2/5s of the cerebral hemisphere

- 1- cranial nuclei
- 2- A tract
- 3- sensory
- 4- centers R.C.

- Each **POSTERIOR CEREBRAL ARTERY** gives the following **branches**:

1. Posterior **Communicating** artery. *anastomosing & post-communicating of carotid*
2. **Capsular** branch supplying the ventral half of the posterior limb of the internal capsule, the thalamus and the geniculate bodies (Thalamo-geniculate artery).
3. **Cortical** branches to the posterior 2/5s of the cerebral hemisphere.

- In its course, the **VERTEBRO-BASILAR system** gives the following **branches**:

- Two **spinal arteries** which unite to form the **anterior spinal artery**.
- Three **cerebellar arteries** on each side: **Superior**, **Middle** & **Inferior**.

SP and cerebellum

all segments of cord bilaterally except PICA

CIRCLE OF WILLIS

FORMATION:

1. **Anteriorly**: by the union of both anterior cerebral arteries through the anterior communicating artery.
2. **Posteriorly**: by the union of each **posterior cerebral artery** with the **ICA** of the same side through the posterior communicating artery.

Therefore, in the circle of Willis:

- The two carotid arteries communicate with each other, and with the vertebro-basilar system.

SITE:

It is present in the subarachnoid space at the base of the brain.

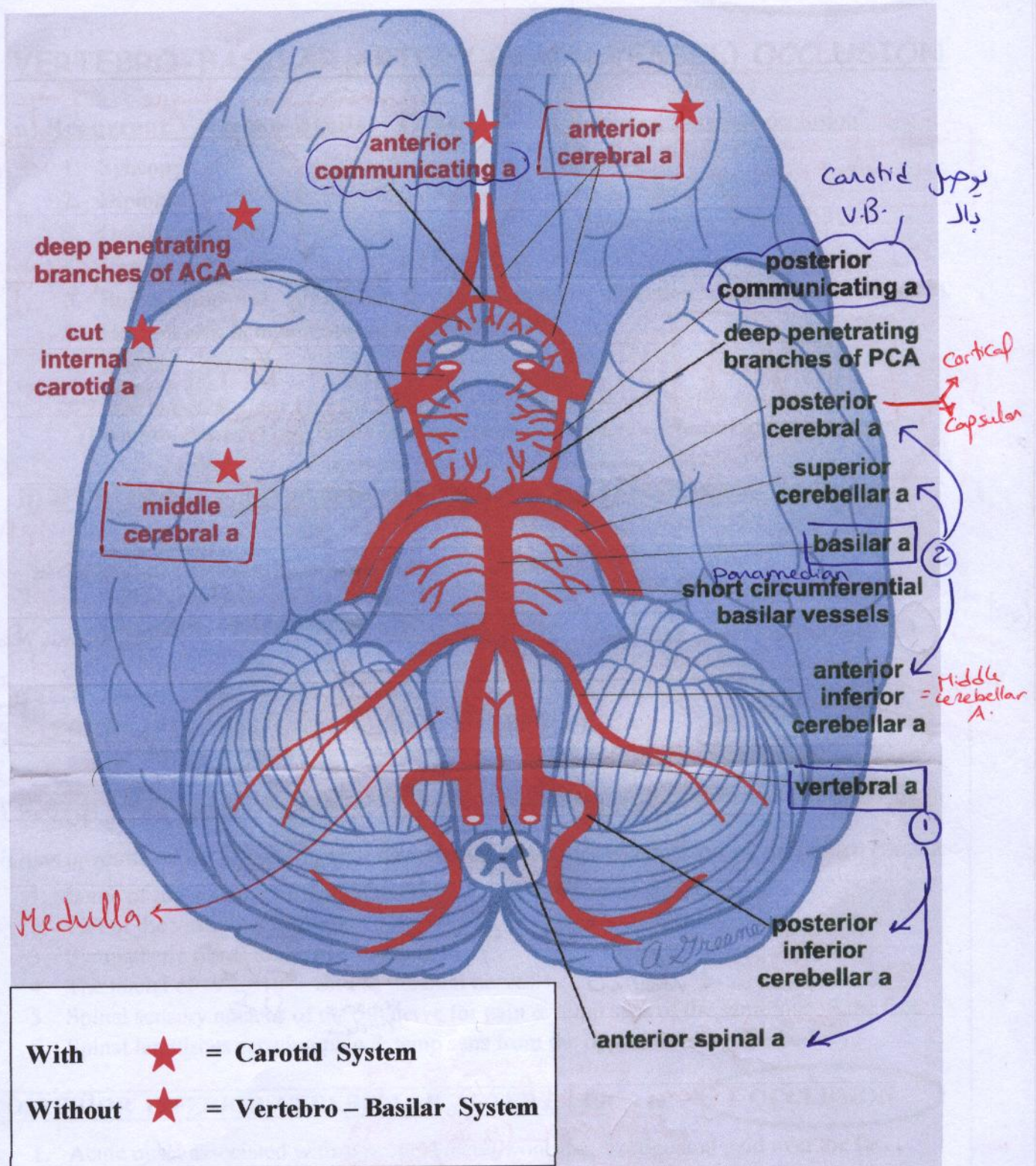
RELATIONS

- **Anteriorly**: to the olfactory tract & optic chiasma.
- **In the middle**: to the cavernous sinus & optic chiasma.
- **Posteriorly**: to the midbrain & oculomotor nerve. 3

only gives posteriorly Ant. lat. s/o, if occluded pyramidal affected



CIRCLE OF WILLIS



II. VERTEBRO-BASILAR SYSTEM

OCCLUSION

VERTEBRO-BASILAR ARTERY (MAIN VESSEL) OCCLUSION

a) Recurrent Vertebro-Basilar TIAs:

$\frac{1}{4} \rightarrow \frac{1}{2}$ hour (< 24 hrs)

"may precede complete occlusion"

RAS

1. Syncope.
2. Diplopia.
3. Ophthalmoplegia.

3, 4, 6

Vestibular N

4. Vertigo or tinnitus.

5. Bulbar symptoms (dysphagia, dysarthria, nasal regurgitation, and hoarseness of voice).

6. Hemiparesis, hemianaesthesia or parasthesias.

7. Ataxia.

"These manifestations indicate insufficiency of the Vertebro-Basilar Artery, (Vertebro-Basilar TIAs) which may terminate in complete occlusion of the artery."

MCC Most common symptom of Vertebro Basilar is Vertigo

9 pharynx

10, 12 tongue, palate

10 Palate

10 vocal cords



b) STROKE (Complete occlusion):

Stage 3 atherosclerosis (closed lumen)

> 24 hours "may be preceded by TIAs"

RAS

- Deep coma.
- Complete quadriplegia with decerebrate rigidity.
- Bulbar paralysis.
- Respiratory embarrassment.

Usually fatal

Both medial & lateral lemnisci (All sensation)

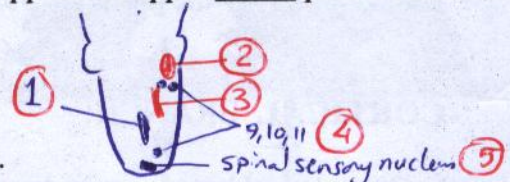
death causes R.F aspiration pneumonia Bulbar

POSTERIOR INFERIOR CEREBELLAR ARTERY (PICA):

= Wallenberg syndrome, lateral Medullary syndrome

ANATOMY

- It arises as a branch of the vertebral artery & terminates to supply the inferior part of cerebellum.
- It runs upwards on the lateral aspect of the medulla & supplies its upper lateral part which contains:
 1. Some of the reticular formation nuclei.
 2. Part of the vestibular nucleus.
 3. Sympathetic fibres to the eye and face.
 4. The nuclei of 9th, 10th & 11th cranial nerves.
 5. Spinal sensory nucleus of the 5th nerve for pain & temp sens of the same side of the face.
 6. Spinal lemniscus carrying pain & temp sens from the opposite side of the body.



POSTERIOR INFERIOR CEREBELLAR ARTERY (PICA):

OCCLUSION

1. Acute onset associated with syncope, hiccup, vomiting, vertigo and pain over the face.
2. Ipsilateral cerebellar ataxia (nystagmus, dysarthria, incoordination).
3. Ipsilateral Horner's syndrome.
4. Ipsilateral palato-pharyngeal-laryngeal paralysis & weakness of sternomastoid & trapezius.
5. Ipsilateral loss of pain and temperature sensations over the face.
6. Contralateral loss of pain and temperature sensations over the body.

Cerebellum

RAS

RC

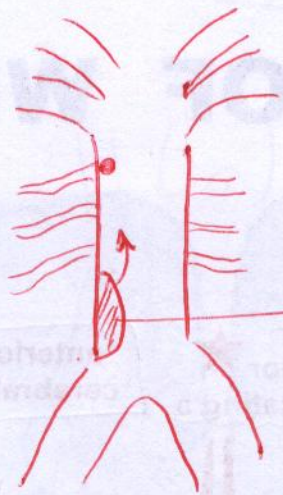
Horner's

Staccato or scanning

vestibular N.

11

LMNL cup nucleus



atheromatous plaque (stage II)
only narrow lumen

either
Rupture or thrombus on top (stage III)

detached parts

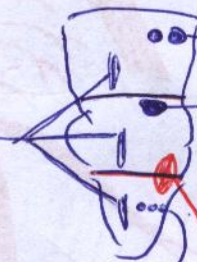
TIA

تروح تيسر ال A.A. ال تروح Brainstem

① syncope

من
Coma
لايقضونه

RAS



② diplopia

③ ophthalmoplegia

شلل عضلات العين
ومقدرة التحريك
لوالعين
لانه لازم يتعاون معي

9, 10, 12

bulbar nuclei

⑤ Bulbar symp.

pharynx
larynx
palate
vocal cord

Coma
ايقضونه

④ vertigo
tinnitus

vestibular nucleus
Coma لو

vestibular nucleus

مع واتزان

motor nucleus
sensory nucleus

VIII
VIII

reflexes palatal ⑩

gag ⑨, ⑩

inspect uvula ⑫

vocal cord ⑩

BRAIN STEM BRANCHES OCCLUSION

Paramedian

"Syndromes of Brain Stem Hemiplegia: Crossed Hemiplegia"

ج62-1

POSTERIOR CEREBRAL ARTERY OCCLUSION

1. CAPSULAR BRANCH (Thalamo-geniculate artery):

ANATOMY

- It supplies: the ventral half of the posterior limb of the internal capsule, the thalamus and the geniculate bodies. *vision ✓ as overlapped by MCA. Auditory ✓ as Bilaterally supplied*

CAPSULAR BRANCH (Thalamo-geniculate artery):

OCCLUSION

★ Thalamic Syndrome:

1. Thalamic pain: severe burning pain on the contralateral side.
2. Reflex dystrophy of the shoulder girdle & arm secondary to the pain.
3. Contralateral hemianaesthesia. *all types of sensations*
4. Extrapyrmidal manifestations: due to ischemia of the basal ganglia.

★ *Contralat. monoplegia in L.L.*

*حرقان
في ذراع
والجمل
↓
ischemia
fibrosis
↓
لا يتحرك
atrophy*

*as frozen
shoulder
syndrome
in MI
(Q/P)*

*eg
Chorea
& parkinsons*

2. CORTICAL BRANCH:

ANATOMY

- It supplies: the posterior 2/5s of the cerebral hemisphere.

*occipital 17, 18, 19
& post part of temporal (receives overlap from MCA)*

CORTICAL BRANCH:

OCCLUSION

1. Contralateral homonymous hemianopia + preserved light reflex + preserved macular vision.
2. Visual agnosia (in lesions of Domin Hemi).

*17
18, 19*

Macula & area 17

*oral
double
MCA*

MAIN ARTERY OCCLUSION

1. Thalamic Syndrome.
2. Contralateral homonymous hemianopia + preserved light reflex + preserved macular vision.
3. Visual agnosia (in lesions of Domin Hemi).
4. *Contralat Monoplegia L.L.*

NEUROGENIC BLADDER

I. LMNL (Lesions at the level of the reflex arc)

- Sensory Atonic Bladder** (Lesion in the afferent sensory fibres)
 - Absence of the sense of fullness of the bladder.
 - Retention of urine with overflow. *لا زلة من حاسس out pain*
- Motor Atonic Bladder** (Lesion in the efferent motor fibres)
 - Preservation of the sense of fullness of the bladder.
 - Retention of urine due to inability to evacuate the bladder voluntarily.
 - Catheterisation is needed. *no overflow (as pain retention)*
- Autonomic Bladder** (Lesion in spinal center S2,3,4 or in both afferent & efferent fibres)
 - Involuntary & Incomplete evacuation of the bladder.
 - Evacuation of the bladder occurs by its myogenic contraction.

II. UMNL (Lesions above the level of the reflex arc)

- Acute:**
 - Retention of urine with overflow.
- Gradual:**
 - Partial lesion: *Bilat but some fibers are preserved* Precipitancy of micturition.
 - Complete lesion: *Automatic Bladder*
 - Complete evacuation of the bladder.
 - Evacuation of the bladder occurs by the spinal reflex arc. (NO control)

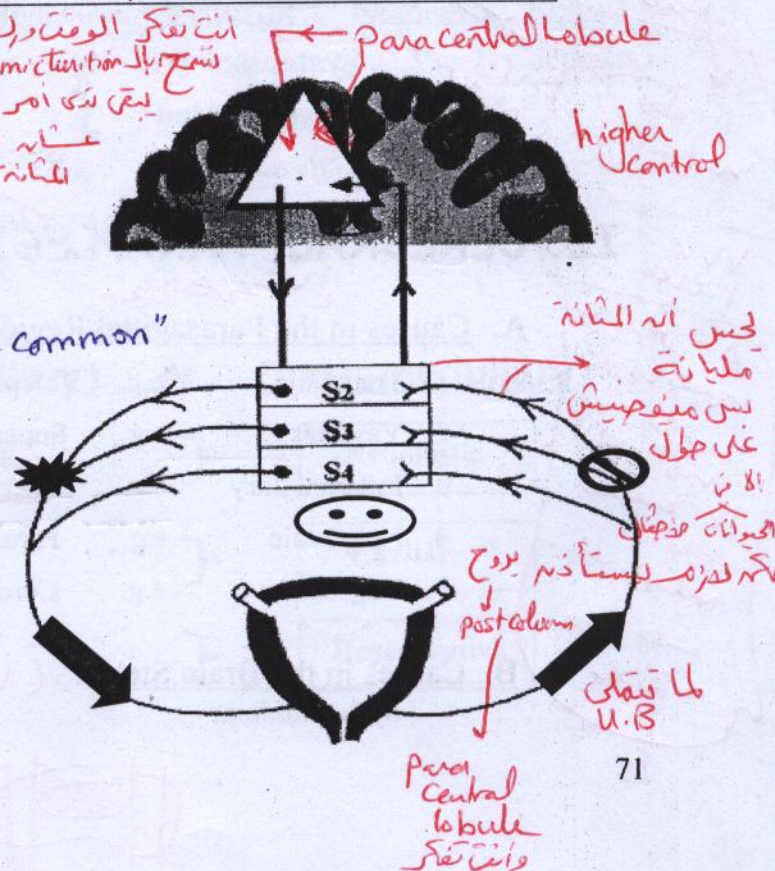
For any disturbance in bladder function to occur, the lesion should be bilateral.

I. UMNL

- Acute:**
 - Retention with overflow.
- Gradual:**
 - Partial: Precipitancy. "more common"
 - Complete: Automatic bladder.

II. LMNL

- Sensory atonic bladder. 
- Motor atonic bladder. 
- Autonomic bladder. 



Long & short clinical

Diagnosis or levelling > نظري

72

PARAPLEGIA

DEFINITION

Paralysis or Paraparesis of both lower limbs.

الذراعين
سلاسل

TYPES

- SPASTIC PARAPLEGIA: due to UMNL (any lesion in the Pyramidal pathway).
- FLACCID PARAPLEGIA: due to LMNL (any lesion from the AHCs till the muscles).

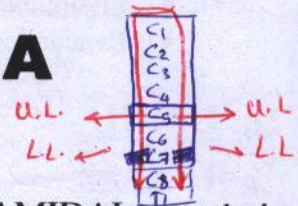
motor power
tone & reflex
ما للناس دعوة بال
Bilat

SPASTIC PARAPLEGIA

DEFINITION

- Paralysis or paresis of both LLs due to BILATERAL PYRAMIDAL tract lesion.

دودة هو الاشر
unilat
Brown sequard



TYPES

L.L. take from L2 → S2 (L2,3,4,5,S1,2)
حالة حيث تحت C5 يبقى مشترك، الجسيم بس انه L.L. يأخذ من C5

I. SPINAL PARAPLEGIA

The most common type

1. Focal:
2. Systemic
3. Disseminated

paraplegia with a level = sensory level
paraplegia without a level



II. CEREBRAL PARAPLEGIA

The least common type

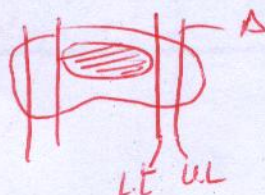
A. Causes in the Parasagittal Region: (area of cortical presentation of LLs)

1. Traumatic e.g. Depressed fracture of skull, Subdural hematoma.
2. Vascular e.g. Superior Sagittal Sinus thrombosis.
3. Inflammatory e.g. Encephalitis, Meningio-encephalitis.
4. Neoplastic e.g. Parasagittal meningioma.
5. Degenerative e.g. Cerebral palsy (Little's disease).

B. Causes in the Brain Stem:

- Syringobulbia.
- Midline brain stem tumours.

(V. late تفريق)
(عالم بالمشاكل)



تفريق الحبل
Bibers
L.L. يتأخر

72

WhiteKnightLove

Acute
Subacute
Chronic

acc. to
onset

acc. to
site
Intra
Extra

73

CAUSES OF SPINAL PARAPLEGIA

1. FOCAL PARAPLEGIA:

due to:

A. Compression:

Acute,

Subacute,

Chronic

1. Acute

hours → days

(extramedullary)

من عظم يضغط
من بركة

- TB of the spine:

Pott's disease.

Inflammatory

- Fracture or fracture-dislocation of the vertebrae.

Traumatic

- Disc prolapse.

weeks → Months

2. Subacute

(extramedullary)

or

(intramedullary)

- TB of the spine:

Pott's disease.

Inflammatory

- Tumours of the spine.

Extra
Intra
vertebrae
roots
meninge.
Ependymoma
Glioma

Neoplastic

TUMOURS THAT COMPRESS THE SPINAL CORD MAY ARISE FROM:

1. Extramedullary:

(Outside the spinal cord: Vertebrae, Meninges, Roots)

- Vertebrae: Extradural: e.g. 1ry osteosarcoma, or 2ry deposits.
- Meninges: Dural: e.g. meningioma.
- Roots: Intradural: e.g. neurofibroma.

2. Intramedullary:

(Inside the spinal cord parenchyma itself)

- Glioma or Ependymoma.

3. Chronic

(years)

(intramedullary)

or

(extramedullary)

- Glioma or Ependymoma.

Neoplastic

- Syringomyelia.

Cavitary

- Spondylosis.

Degenerative

as disc prolapse
acute

73

B. Inflammatory:

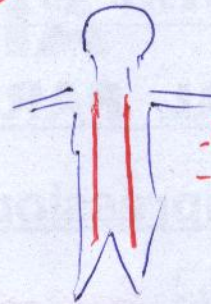
- Transverse myelitis.

C. Vascular:

- Anterior spinal artery occlusion.

ASA from vertebral
doesn't give post column
sensation

Oral
Paraplegia
sensation
1. Vascular
e.



- Bilat
- symmetrical

2. SYSTEMIC PARAPLEGIA:

due to:

- A systemic disease affects one or more systems selectively & is usually bilateral & symmetrical.
- A systemic disease affects the pyramidal tracts, either alone or with other tracts → paraplegia.

A. Hereditary:

1. Hereditary spastic paraplegia.
2. Hereditary ataxias, e.g. Friedreich's ataxia or Marie's ataxia.

B. Secondary:

1. Pellagra lateral sclerosis. B complex
2. Subacute combined degeneration (SCD). B12

Para or Quadri

PN
Post column
Pyramidal tract

no level

C. Idiopathic:

- Motor neurone disease (MND). → selective on Δ only (or) selective on AHs

3. DISSEMINATED PARAPLEGIA:

due to: demyelination

- A disseminated disease is a multifocal disease of the same nature:

1. Disseminated Sclerosis (DS): "Refer to DS".
2. Disseminated Encephalo-myelitis (DEM).
Fever, headache, coma

قسط DS



Patchy

DIAGNOSIS OF SPASTIC PARAPLEGIA

I. CLINICAL DIAGNOSIS

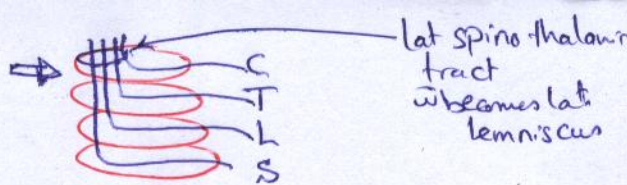
Criteria to diagnose Spastic Paraplegia

1. Paralysis or paresis of both LLs.
2. Hypertonia (clasp knife spasticity).
3. Hyper-reflexia (and may be clonus).
4. Positive Babinski sign.
5. Sphincteric troubles, especially precipitancy of micturition.

or Autonomic

if partial
if complete

extra medullary
fibers of
sacral are
most lateral
يعتبر على مستوى



Intra medullary

SLT

at cervical C5 C6
75
cervical & thoracic
SLT

at thoracic C6 C7

II. ANATOMICAL DIAGNOSIS

1 Is it: Systemic, Disseminated or Focal paraplegia ??

- **Systemic:** Paraplegia without a level
 - Onset: Gradual Course: Progressive.
 - Affection: Symmetrical affection.
- **Disseminated:** Paraplegia without a level
 - Onset: Acute Course: Remissions & exacerbations.
 - Affection: Multiple nervous system affection (dissemination).
- **Focal:** Paraplegia with a level
 - Below the level: * UMN manifestations & Sensory loss.
 - At the level: * LMN manifestations & Radicular manifestations.

2 Clinical Picture of Focal Paraplegia:

1. Vertebral manifestations:

- Localised pain & tenderness.
- Localised swelling & deformity.

2. Radicular manifestations:

a) Posterior root affection:

- Early: Girdle pain: back pain referred to the distribution of the affected root.
- Late: Sensory loss: in the dermatome supplied by the affected root.

b) Anterior root affection:

- Localised LMN weakness in the muscles supplied by the affected root.

3. Cord manifestations:

a- Sensory affection:

Extra-medullary lesions

Sensory level: below which all types of sensations are diminished.
Sacral affection: early loss of sensations in the saddle area (S3,4,5).

Intra-medullary lesions

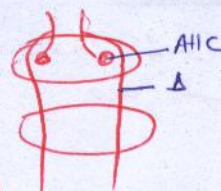
Jacket sensory loss: of dissociated nature (lost pain & temp. & preserved touch & deep).
Sacral spare: preserved sensations in the saddle area (S3,4,5).

b- Motor affection:

- LMNL (with fasciculations): at the level of the lesion due to affection of the AHCs.
- UMNL: below the level of the lesion due to affection of the descending pyramidal tracts.

c- Sphincteric affection:

- Early in Intra-medullary & Late in Extra-medullary lesions
- Acute lesions: Retention with overflow.
- Gradual lesions: Precipitancy (partial lesion), then: Automatic bladder (complete lesion).



Extra medulla
AHC
منطقة



White Knight Love

3 Is it: Extra-medullary or Intra-medullary paraplegia??

- Refer to the table.

III. DIAGNOSIS OF STAGE OF PARAPLEGIA

1. Stage of flaccid paralysis (shock stage)

- It occurs in acute lesions & lasts from 2-6 weeks.
- It is characterized by:

- o Paralysis of both LLs.
- o Hypotonia & Hyporeflexia.
- o No plantar response.
- o Retention of urine with overflow.

as if LMNL

acute compression causes inflammatory vascular

<i>spasticity</i> Δ		<i>ED rigidity</i>
extensors	L.L. reflex	flexors
hyperreflexia		hyporeflexia

2. Stage of spastic paralysis (established paraplegia)

- It occurs in: a) Recovery of acute lesions, or b) Gradual lesions from the start.
- It passes into 2 stages:

A. Paraplegia in Extension:

- It occurs in INCOMPLETE injury of the spinal cord, where there is affection of the pyramidal & sparing of the extra-pyramidal tracts.
- It is characterized by:
 - Hypertonia (spasticity): affects the extensors more than the flexors. *Δ antigravity*
 - Deep reflexes: exaggerated & may be CLONUS.
 - PRECIPITANCY of micturition. *(partial lesion)*

B. Paraplegia in Flexion:

- It occurs in COMPLETE transection of the spinal cord, where there is affection of both pyramidal & extra-pyramidal tracts.
- It is characterized by:
 - Hypertonia (spasticity): affects the flexors more than the extensors.
 - Deep reflexes: less exaggerated & NO CLONUS.
 - AUTOMATIC BLADDER. *(complete)*
 - Positive mass reflex:

Any stimulus below the level of the lesion, results in:
withdrawal of the legs, sweating below the level,
micturition, defecation, & ejaculation.

partial & complete spinal cord injury

precipitous

Δ reflex ED reflex

denervation supersensitivity neurones $\rightarrow$ stimulus

unexplained

	Paraplegia in extension	Paraplegia in flexion
1. Cause	Pyramidal lesion	Pyramidal & extrapyramidal
2. Hyypertonia	More in extensors	More in flexors
3. Position of LLs	Extended	Flexed
4. Deep reflexes	Exaggerated	Less exaggerated
5. Clonus	Present	Absent
6. Mass reflex	Absent	May be present
7. Bladder	Precipitancy	Automatic bladder

IV. ETIOLOGICAL DIAGNOSIS

- Differential Diagnosis of the causes.

INVESTIGATIONS

I CSF

Formation & circulation:

- Normally the CSF is a clear colourless fluid, formed in the cerebral ventricles.
- It leaves the ventricles through the lateral and medial foramina of the fourth ventricle to the subarachnoid space covering the brain & the spinal cord.
- It is then finally absorbed into the blood to internal jugular veins.

Functions:

- Shock absorption: it acts as a **cushion** for the CNS and protects it from traumatic injury.
- Nutrition: it contains sugars & other elements that are used by the CNS cells.
- Waste disposal: it removes waste products produced by metabolism of CNS cells.

Absorption of the CSF from the subarachnoid space into the blood stream takes place through the **arachnoid villi**. When the CSF pressure is greater than the venous pressure, CSF will flow into the blood stream. However, the arachnoid villi act as "**one way valves**" ...if the CSF pressure is less than the venous pressure, the arachnoid villi will **NOT** let blood pass into the ventricular system.

	Paraplegia in extension	Paraplegia in flexion
1. Cause	Pyramidal lesion	Pyramidal & extrapyramidal
2. Hypertonia	More in extensors	More in flexors
3. Position of LLs	Extended	Flexed
4. Deep reflexes	Exaggerated	Less exaggerated
5. Clonus	Present	Absent
6. Mass reflex	Absent	May be present
7. Bladder	Precipitancy	Automatic bladder

IV. ETIOLOGICAL DIAGNOSIS

- Differential Diagnosis of the causes.

INVESTIGATIONS

I

CSF

کمر نیسالی
فالیسکوی

ما یلش قیقه کیری
invasive
و کمان

استرج
کلی استرج
paraplegia
کتاب

استرج
کلی استرج
- DS
- HND or SCD
Focus - ASA : کمر نیسالی
- Transverse myelitis : کمر نیسالی
- Pott's : کمر نیسالی

Formation & circulation:

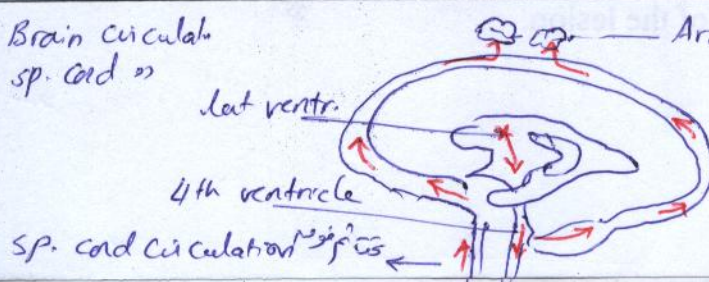
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- 1st Brain circulation
- then sp. cord



Arachnoid villi
= project
= granulation

begin at arachnoid
& project
in dura to end in
dural venous sinuses
(Blood)

1) CSF pressure:

It is measured by tapping the CSF in the lumbar region, using a needle connected to a spinal manometer.

- Normally it is 120 – 140 mmH₂O.
- In extra-medullary compression: it is markedly diminished.
- In intra-medullary compression: it is moderately diminished.

2) Queckenstedt's test:

“UNRELIABLE TEST”

- Bilateral jugular vein compression normally ↓ the flow of the CSF from the SAS to the blood & leads to rapid rise of CSF pressure in the manometer. Release of the compression leads to rapid drop of pressure to normal.
- In Extra-medullary Compression: (complete dynamic block)
There is no change of CSF pressure, due to complete obstruction of the SAS.
- In Intra-medullary compression: (partial dynamic block)
There is a slower & lesser rise of pressure, due to incomplete obstruction of the SAS.

3) CSF contents:

Normal CSF contents

clear & colorless

- Proteins: 20 - 40 mg / 100 ml.
- Cells: 0 - 5 / HPF., mainly lymphocytes.
- Chlorides: 720 - 750 mg / 100ml.
- Sugar: 50 - 80 mg / 100 ml.

- In Extra-medullary compression:

There is marked increase in proteins leading to: “Froin's Syndrome”

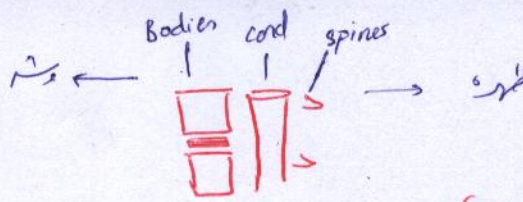
- Spontaneous coagulation. → as from proteins that diffuse to CSF there are fibrin & fibrinogen to cause coagulation
- Xanthochromia (yellowish discoloration).
- Cyto-albuminous dissociation (↑ proteins BUT near-normal cell count).

- In Intra-medullary compression:

There is no marked change.

2 CT scan & MRI

- Accurate in localization of the lesion.



(Extramedullary)

79

3 Plain X-Ray of the Spine

"in vertebral causes only"

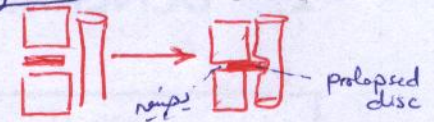
- Destruction of the vertebrae: Pott's disease, Trauma or Tumours.
- Narrowing of intervertebral spaces $\pm$ osteophytes: Spondylosis & acute disc prolapse.

= calcification
degenerative
only
spondylosis
not
Acute prolapse

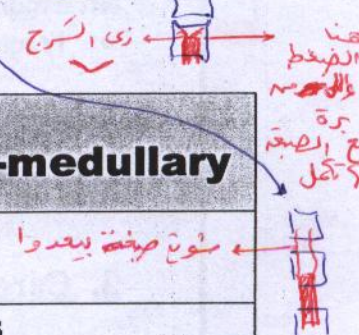
4 Myelography

A radio-opaque substance is injected into the SAS and then X-ray pictures are taken:

- Extra-medullary compression: **= saddle shaped** complete block with short tails.
- Intra-medullary compression: $\uparrow$ width of the cord with long tails.



	Extra-medullary	Intra-medullary
HISTORY		
Onset	Painful	Painless
Sphincteric affection	Late or Absent	Early
CLINICAL PICTURE		
Sensory	Sensory level: below which all types of sensations are diminished. Sacral affection: early loss of sensations in the saddle area (S3,4,5).	Jacket sensory loss: of dissociated nature (lost pain & temp. & preserved touch & deep). Sacral spare: preserved sensations in the saddle area (S3,4,5).
Sphincteric affection	Late or Absent	Early
INVESTIGATIONS		
CSF	Marked $\downarrow$ of pressure. Complete dynamic block. Froin's syndrome.	Moderate $\downarrow$ of pressure. Partial dynamic block. No Froin's syndrome.
CT & MRI	Diagnostic	Diagnostic
Plain X-Ray	Possible vertebral lesion	Normal
Myelography	Saddle-shaped block	Fusiform-shaped block



79

TREATMENT

I. GENERAL CARE

1. Care of the skin:

- Frequent change of: the patient's position (every 2 hours), and of the bed sheets.
- Frequent wash of: skin of the back & pressure points by alcohol followed by powder.

2. Care of nutrition & fluid balance:

- Tube feeding & IV fluids.

3. Care of the bladder:

- Catheterisation & urinary antiseptics.
- Parasympathomimetics: in case of retention.

4. Care of the bowel:

- Daily enema.

II. SYMPTOMATIC

1. Vitamin and tonics.
2. Muscle relaxants.

III. PHYSIOTHERAPY.

IV. SPECIFIC:

"treatment of the cause"

e.g. Anti-tuberculous drugs in case of Pott's disease.

SPECIAL FORMS OF PARAPLEGIA

Oral loss infection

TRANSVERSE MYELITIS

DEFINITION

It is an ACUTE INFLAMMATION affecting the **gray & white** matter in one or more adjacent spinal cord segments; commonly the thoracic segments.

ETIOLOGY

1. **IDIOPATHIC.** → mostly autoimmune as responds to cortisol

2. **INFECTION:**

- **Viral:** Nonspecific, Influenza, Chicken pox.
- **Bacterial:** TB, Syphilis, Diphtheria.

3. **IMMUNOLOGIC:**

- Following vaccination. (esp. of rabies)
- Associated with vasculitis: e.g. SLE. Abs against cord

4. **IATROGENIC & TOXIC:**

- Post lumbar. after injection in spinal anesthesia → irritation & inflammation of sp. cord
- IV heroin. / Amphetamin

5. **DEMYELINATING:**

DS & DEM.

Oral

disseminated or the cord as
Paraplegia patchy cord level

CLINICAL PICTURE

Shock stage:

استرجاع كامل

(2 - 6 weeks)

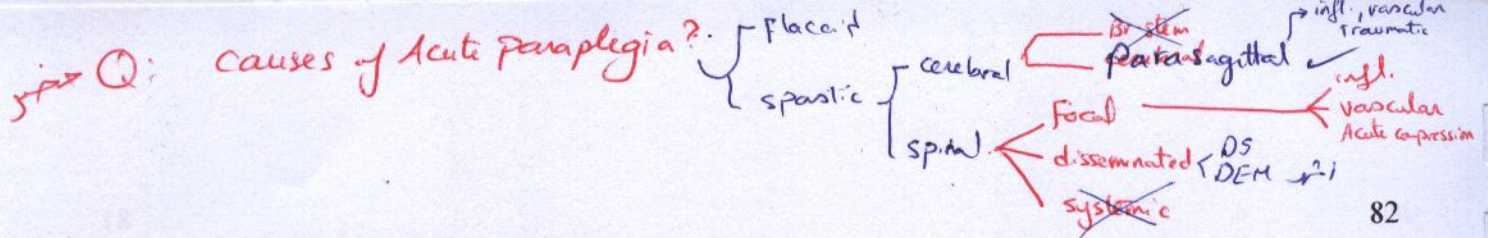
فocal
TM وده من مشهور
The shorter duration the better prognosis

- Paraplegia: Flaccid paraplegia + sensory level below which all types of sensations are lost.
- Sphincteric: Retention of urine with overflow.

Recovery stage:

استرجاع كامل

- Paraplegia: Spastic paraplegia + sensory level below which all types of sensations are lost.
- Sphincteric: Precipitancy of micturition.



DIFFERENTIAL DIAGNOSIS

- Acute cord compression.
- Anterior spinal artery occlusion.
- Guillain-Barré syndrome.

INVESTIGATIONS

CSF examination:

- o Increased both cells and proteins.
- o In DS there is high immunoglobulin level.

MRI:

- o To exclude acute cord compression.

TREATMENT

1. Nonspecific symptomatic tt.
2. CORTICOSTEROIDS:

- Prednisone (1 mg / Kg / day, orally): in: Idiopathic cases, SLE, DS.
- ACTH (IM): in: DS in acute exacerbation

ANTERIOR SPINAL ARTERY OCCLUSION

ETIOLOGY

Thrombosis, Embolism, Dissecting aortic aneurysm.

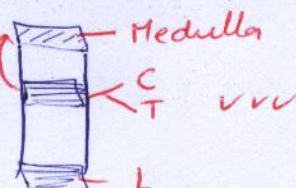
CLINICAL PICTURE

Similar to Transverse Myelitis but there is sparing of the deep sensations because the posterior columns of the spinal cord are supplied by the posterior spinal artery.

DEFINITION

It is a chronic disorder characterised by:

1. **Lesion:** Long CAVITIES surrounded by GLIOSIS.
2. **Site:** In the CENTRAL part of the gray matter of the spinal cord:
 - Most commonly: in the lower cervical & upper thoracic segments.
 - Less commonly: in the lumbar segments OR may extend up to the medulla (Syringobulbia).



ETIOLOGY

1. Congenital.

2. Idiopathic.

CLINICAL PICTURE

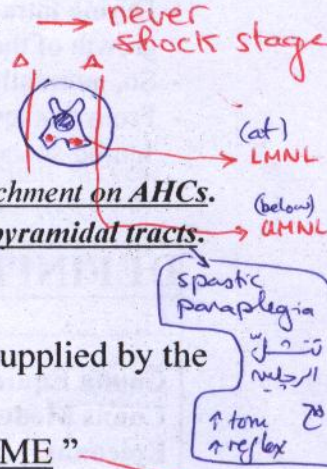
- Age: Commonly between 15 & 35 years.
- Onset and course: Gradual onset & slowly progressive course.

I. Cervical Syringomyelia [Focal Intra medullary]

1. Motor manifestations:

At
Below

- **Early:** In UL: Localised LMN weakness + fasciculations due to encroachment on AHCs.
- **Late:** In LL: Spastic paraplegia (UMN) due to encroachment on the pyramidal tracts.



2. Sensory manifestations:

1. **Jacket sensory loss of dissociated nature** in the area of skin supplied by the affected segments with sacral spare.
2. **preserved deep**

3. Autonomic manifestations: VC due to Parasymp "MORVAN'S SYNDROME"

- Trophic ulcers of the fingers, painless nail infection & vasomotor changes.
- **Horner's Syndrome** (ptosis, myosis, enophthalmos, anhidrosis) (coldness + peripheral cyanosis)
- Associated skeletal anomalies: pes cavus, & spina bifida.
- sphincteric troubles: Early (as intramedullary)

II. Lumbar Syringomyelia:

Picture of: **epiconus lesion** (see later).

precipitancy or automatic (as gradual)

INVESTIGATIONS

- CT scan.

MRI for

also
Haller
PICA
pancast

- Myelography.

TREATMENT

1. X-ray irradiation of the affected region of the cord.
2. Physiotherapy & symptomatic treatment.

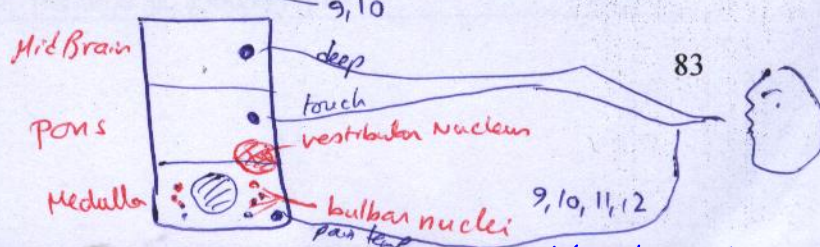
SYRINGOBULBIA

- It is a lesion in the MEDULLA similar to that in syringomyelia.
- The lesion may start in the medulla OR may be an upward extension of a cervical syringomyelia.
- It involves: the spinal nucleus of 5th cranial nerve, Bulbar nuclei & Vestibular nucleus.

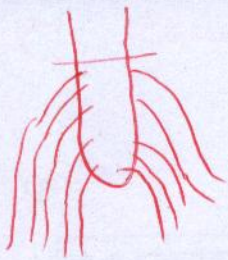
CLINICAL PICTURE

- PAIN IN THE FACE followed by loss of sensation of dissociated nature.
- BULBAR SYMPTOMS with lost palatal and pharyngeal reflexes + **wasting of the tongue.**
- VERTIGO.

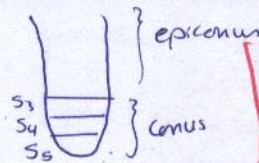
dysarthria
dysphagia
hoarseness voice
Regurgitation



WhiteKnightLove



Lumbosacral roots
جذور الحبل



نظري
clinical
مؤكّد

84

CAUDA EQUINA

= Flaccid paraplegia

علايه
حالات في
flaccid paraplegia

ANATOMY

- During intra-uterine life, the rate of growth of the vertebral column is faster than the rate of growth of the spinal cord.
- So, normally the spinal cord ends at the junction of L1 & L2 vertebrae.
- From this level downwards, the spinal canal is not empty; it is occupied by the collection of lumbo-sacral roots, known as the "**Cauda Equina**."

DEFINITIONS

- Cauda Equina** = collection of lumbo-sacral **roots** in the lower part of the spinal canal.
Conus Medullaris = the lowermost 3 segments of the **spinal cord**: S3,4,5.
Epiconus = the 4 segments of the **spinal cord** above the conus medullaris: L4,5 S1,2.

CAUDA EQUINA LESION

ETIOLOGY

"bone vertebrae"

- Fracture or fracture-dislocation of the lumbar vertebra. }
- Disc prolapse. } *Acute prolapse*
degenerative (spondylosis)
- TB of the spine: Pott's disease. (not common here) }
but the most common to be M.T. therapeutic
- Neoplastic diseases of the spine: }
 - Primary: e.g. osteosarcoma.
 - Secondary (metastatic) from a primary carcinoma.
- Lumbar spondylosis. }
- Spina bifida. }

Traumatic

Inflammatory

Neoplastic

Degenerative

Congenital

84

* usually unilateral → roots ^{roots} _{roots}
 & if Bilat → asymm

CLINICAL PICTURE

I. MOTOR MANIFESTATIONS

- Paralysis or weakness of LMN nature in the LL (in one or both LLs).
- The distribution of weakness depends on the affected roots.

The affection is usually unilateral, **BUT**: if bilateral, it is asymmetrical

II. SENSORY MANIFESTATIONS

- *Early: Radicular pain:* in the distribution of the femoral or sciatic nerves.
- *Late: Radicular sensory loss:* the distribution depends on the affected roots.

The affection is usually unilateral, **BUT**: if bilateral, it is asymmetrical

III. AUTONOMIC MANIFESTATIONS

1. Sphincteric manifestations:
 - They occur if the lesion is **bilateral** & affects **S 2,3,4** roots.
 - They are in the form of:
 - a) Sensory atonic bladder, or
 - b) Motor atonic bladder, or
 - c) Autonomic bladder.
2. Trophic changes.

Distribution of Motor Affection

Root	Action	Muscles
L2	Flexion of the hip	Ileopsoas.
L3	Extension of the Knee	Quadriceps
L4	Dorsiflexion of the ankle	Anterior tibial group
L5	Dorsiflexion of the toes	Anterior tibial group
S1	Plantar flexion of the ankle & toes	Calf muscles
S2	Flexion of the knee	Hamstrings
S3,4,5	Anal contraction	Anal & perianal muscles

of saddle area

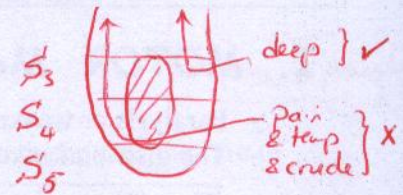
Distribution of Sensory Affection

Root	Sensory supply
L1	Upper third of the front of the thigh.
L2	Middle third of the front of the thigh.
L3	Lower third of the front of the thigh.
L4	Antero-lateral aspect of thigh, Antero-medial aspect of leg, Medial aspect of dorsum of foot, & Big toe.
L5	Lateral aspect of the thigh, Middle third of the dorsum of the foot, & Middle 3 toes.
S1	Postero-lateral aspect of the thigh & leg, Lateral third of the dorsum of the foot, & Little toe.
S2	Posterior aspect of the: Thigh & Leg & Foot.
S3,4,5	Saddle area: Anal, perianal & gluteal region.

CONUS MEDULLARIS LESION

ETIOLOGY

- Causes of Intramedullary compression.



CLINICAL PICTURE

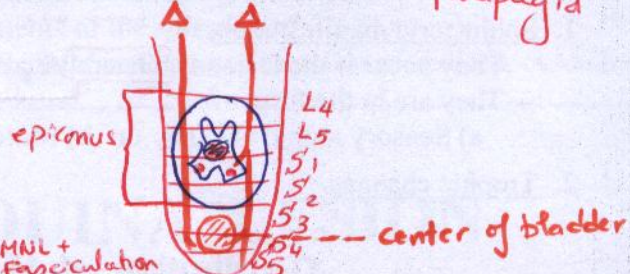
1. Early urinary incontinence (autonomic bladder), & stool incontinence.
2. Impotence. *Loss of erection (parasymp)*
3. Sensory loss in the saddle area, (usually of a dissociated nature).
4. **No** motor or sensory affection in the LLs. *as S3,4,5 not supply*

في حاجية
L.L. من
بال
Paraplegia

EPICONUS LESION

ETIOLOGY

- Causes of Intramedullary compression.



CLINICAL PICTURE

1. **Motor:** *1st: pressure on AHC → LMNL + fasciculation*
then: pressure on Atract → LMNL of Bladder center.
 - Paralysis or weakness in the LLs of LMN nature, in the muscles supplied by L4,5 & S1,2.
Paraplegia (flaccid)

2. Sensory:

- as intramedullary* Sensory loss in the dermatomes supplied by L4,5 & S1,2 (usually of a dissociated nature).

3. Autonomic:

- Precipitancy of urine.
or automatic

الشرج من البول

deep preserved
& fine
as in post-column

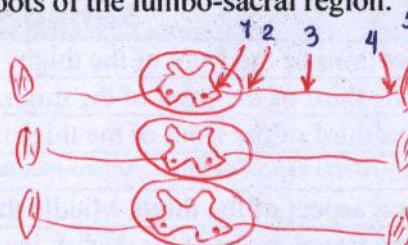
FLACCID PARAPLEGIA

ETIOLOGY

1. Epiconus lesion: *MND* lesion at the AHCs of the lumbo-sacral region.
2. Cauda Equina lesion: lesion at the roots of the lumbo-sacral region.
3. Peripheral nerve lesion. (GBS)
4. NMJ lesion. *Myasthenia*
5. Muscle lesion. *Myopathy*
6. Shock stage of spastic paraplegia.

DIAGNOSIS

1. **Clinical:** LL paralysis of LMN nature.
2. **Anatomical.**
3. **Etiological.**



سؤال ب. ج. د.
معرفة جاذبية حركة
Causes
or
Diagnosis

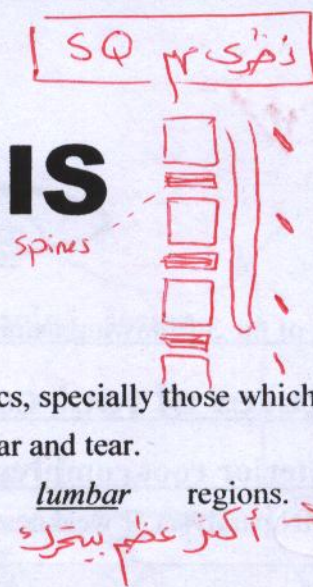
- كيف يجب ان يكون*
- Discuss:**
1. Criteria of flaccid (LMNL مع LMNL)
 2. anatomical diagnosis: discuss C/P of 1, 2, 3 (general C/P motor plus), 4, 5, 6
 3. etiological " : eg GBS

SPONDYLOSIS

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DEFINITION

- It is the gradual, progressive degeneration of the intervertebral discs, specially those which are freely mobile as they are more subjected to the process of wear and tear.
- The freely mobile discs are mainly found in the cervical and lumbar regions.



PREDISPOSING FACTORS

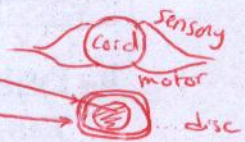
- 1) Old age.
- 2) Occupational: *repeated trauma of the spine in* **LABOURERS** *carrying excess loads.*
- 3) Diabetes mellitus.

4) obesity

بالذات لو عنده كذا حاجة دول

PATHOLOGY

- The intervertebral disc is formed of a central gelatinous part, "**the nucleus pulposus**," surrounded by a fibrous tissue ring, "**the annulus fibrosus**".



DISC DEGENERATION

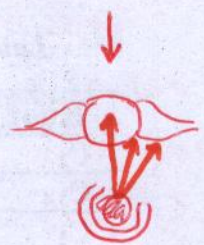
- There is degeneration of the annulus fibrosus → herniation of the nucleus pulposus. This leads to **COMPRESSION** of adjacent structures.
- Since the weakest parts of the annulus fibrosus are the lateral and posterior parts, the herniation will be either: lateral, posterior or posterolateral.

U or Rt

roots

cord

cord & roots



ASSOCIATED DEGENERATION

- Sclerosis of the adjacent surfaces of the vertebrae.
- Lipping or osteophyte formation due to calcification of the prolapsed parts and ligaments.

sclerosis

lipping
زقري



CLINICAL PICTURE

I. CP OF CERVICAL SPONDYLOSIS

IMPORTANT SIGNS

- Hoffman sign: reflex contraction of the thumb & index after nipping the middle finger. *must be lesion above C7 (as U.L. is supplied by C7&T1)*
- L'hermite sign: Electric-like sensation felt in the back & limbs on bending the neck. is due to posterior column affection in the cervical region.

disc → presses on 2 Δ tracts

UMNL

pathological reflexes

cord
LMNL
منافس

also
in DS
بعض الحالات
منافس

also jaw reflex must be
lesion above pons & must be
as its center is C5 w has double Δ supply

White Knight Love 87



PRESENTATIONS

Any one of the 3 following manifestations depending on the direction of prolapse of the disc:

1) Features of root compression

(lateral prolapse)

a. Anterior root compression:

- LMN paralysis or weakness in the muscles supplied by the compressed root.

Root	Action	Muscle
C _{1,2}	Lateral movement of neck	Sternomastoid & trapezius
C _{3,4}	Elevation of shoulder	Supra and infraspinatus
C ₅	Abduction of shoulder	Deltoid
C _{5,6}	Flexion of elbow	Biceps & brachioradialis
C _{6,7}	Extension of elbow	Triceps
C _{7,8}	Extension of wrist	Extensors of wrist
C ₈ , T ₁	Flexion of wrist & movement of small muscles of the hands	Flexors of wrist

b. Posterior root compression:

وجع في الذراع

- **Early:** pain and paraesthesias, due to posterior root irritation, which are referred to the shoulder and ULs (brachial neuralgia).
- **Late:** hyposthesia in the dermatomes supplied by the compressed root.

Root	Sensory distribution
C ₂	Lateral aspect of neck
C _{3,4}	Shoulder down to manubrium anteriorly
C ₅	Lateral aspect of arm
C ₆	Lateral aspect of forearm, thenar eminence & thumb
C ₇	Middle aspect of forearm, middle of palm, middle 3 fingers
C ₈	Medial aspect of forearm, hypothenar eminence & little finger
T ₁	Medial aspect of arm

2) Features of cord compression:

(of extra medullary)

(Posterior prolapse)

a. Compression of the pyramidal tracts:

- Weakness or paralysis: with signs of UMNL below the level of compression.
- Bladder disturbances: rare & late & occur only in severe lesions.

b. Compression of the spino-thalamic tracts:

- Superficial sensory loss below the level of compression.

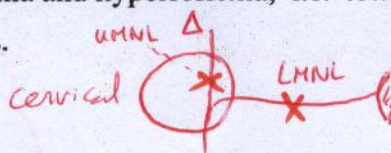
c. Compression of the posterior column:

- Deep sensory loss i.e. sensory ataxia below the level of compression.

3) Features of root & cord compression: (Postero-lateral prolapse)

- a. **In ULs:** combined signs of LMNL in the form of wasting of muscles + signs of UMNL in the form of hypertonia and hyperreflexia, i.e. **tonic atrophy**.

- b. **In LLs:** weakness with signs of UMNL.



Postero lat →

1. UMN : below level : spastic paraplegia
 2. Combined UMN & LMN : at level of lesion →

Tonic ↑ tone ↑ reflex	atrophy wasting
-----------------------------	--------------------

Combined

مع انه المخصوص انه يتوقع
 UMN & LMN
 على نفس المستوى
 يظهر في شكل
 LMN
 لأنه خلا من عصب
 وحب وامل
 أصلا

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II. CP OF LUMBAR SPONDYLOSIS

- This will present with the same picture of Cauda equina lesion.

INVESTIGATIONS

1. MRI & CT scan:

- MRI is the imaging of choice.
- Will detect small disc prolapses missed by other methods of imaging.

2. Plain X-ray:

- Narrowing of the intervertebral disc spaces.
- Sclerosis of the adjacent surfaces of the vertebrae.
- Lipping or osteophyte formation.
- Straightening of the spines (loss of lordosis).

3. Myelography:

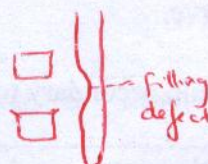
- Anterior filling defect due to disc protrusions and compression of the cord.

Obsolete

in lat. view

as disc prolapse cause
 reflex spasm in para vertebral
 muscles → straighten the column

disc protrusion



filling defect

relief pain
 decrease muscle spasm
 decrease joint stiffness

e.g. NSAIDs

- Analgesics:
- Muscle relaxants. (↓ MS spasm reflex)

II. Physiotherapy:

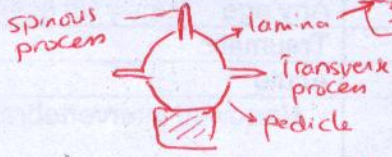
- Short wave therapy. → produce local heat → ++ Blood Flow
- Plastic neck collar: → to ↓ movements
- Traction of the vertebrae. → maximum duration of 3 months to avoid muscle fibrosis. relieve compression on cord symptomatically

III. Surgical:

"Decompression by laminectomy"

INDICATIONS:

1. Failure of the medical treatment.
2. Severe intolerable pain.
3. Sensory or motor affection. sensory loss or wasting
4. Bladder disturbances: rare & late & occur only in severe lesions.



decompression

SCIATICA

فقط احفظ
Causes

DEFINITION

It is radicular pain along the distribution of the sciatic nerve ($L_{4,5}$ $S_{1,2,3}$) i.e. along the back of the thigh, leg and foot.

و طائر دعوة
بالباقى

ETIOLOGY

I. In the spinal canal:

“ at the lumbosacral regions ”

- 1) Acute lumbar disc prolapse.
- 2) Lumbar spondylosis.
- 3) Fracture or dislocation.
- 4) Pott's disease or tumours.

II. In the intervertebral foramina:

- 1) Neurofibroma.
- 2) Radiculitis.

III. In the pelvis:

“ compression of sciatic plexus over the sacro-iliac joint by ”

- 1) Malignant tumours: bladder, rectum, ovaries.
- 2) Pelvic abscess.
- 3) Pregnant retroverted uterus.

IV. In the sciatic nerve:

- 1) Neuritis as diabetic, alcoholic.
- 2) Pressure on the nerve by dislocated head of femur.
- 3) Wrong injection into the nerve.

V. Referred:

sciatic pain secondary to hip or sacro-iliac joint disease.

The most common causes of sciatica are: • Acute disc prolapse • Lumbar spondylosis

Acute disc prolapse:

- There is sudden rupture of the annulus fibrosis followed by bulging (herniation) of the nucleus pulposus; this compresses the spinal roots.
- It may occur at any age and usually follows trauma, as lifting heavy objects.
- Plain x-ray of the back: narrowing of the intervertebral spaces with no degenerative changes.

	Acute disc prolapse	Lumbar spondylosis
Age	Any age	Middle and old age
Cause	Traumatic	Degenerative
Onset	Acute	Gradual
X-ray	- Narrowed intervertebral space	- Narrowed intervertebral space - Sclerosis, lipping & osteophytes

CLINICAL PICTURE

Symptoms

Pain and paraesthesias along the course of the sciatic nerve.

Pain is aggravated by:

1. Walking which stretches the nerve.
2. Coughing, straining or sneezing.

Pain is relieved by:

- Bed rest on a hard mattress in cases of disc lesions.

Signs

- 1) Sensory: Tenderness and discomfort on direct pressure on the sciatic nerve.
- 2) Motor: Slight LMN weakness in the muscles supplied by the nerve and the ankle reflex may be weak or lost.
- 3) Back signs: The paravertebral muscles are spastic; resulting in loss of lumbar lordosis, tenderness and limitation of movements of the spine.
- 4) Signs of meningeal irritation: positive Kernig, Lassegue & Brudzinski signs.

INVESTIGATIONS

- 1) In cases with suspected lesion of the spinal canal:
 - Plain x-ray - Myelography - CT scan & MRI.
- 2) Rectal and vaginal examination.
- 3) Plain x-ray for the hip joint.
- 4) Urine analysis and blood sugar curve.

TREATMENT

1. Treatment of the cause.
2. In cases of disc prolapse:
 - Bed rest on a hard mattress for 2-3 weeks.
 - Analgesics (e.g. NSAIDs) and muscle relaxants to relieve the pain.
 - Physiotherapy.
 - Decompression laminectomy when there is:
 - Failure of medical treatment.
 - Frequent recurrence of pain.
 - Development of a neurological deficit (sensory or motor).

MOTOR NEURONE DISEASE

DEFINITION

- It is a systemic disease affecting the **Motor System only**.
- It may affect the: UMN, or the LMN or both.
- It is a degenerative disease of a gradual onset and progressive course.

Etiology: idiopathic but may be overproduction of free radicals (oxidative stress)

CLINICAL PICTURE

- Age of onset: middle and old ages.
- Sex: males are more affected than females.
- Onset and Course: gradual onset and progressive course.
- Signs and symptoms:
 - o They are usually bilateral (because it is a systemic disease).
 - o They depend on whether the UMN the LMN or both are affected.

Symmetrical

Flaccid & spastic
Clinical Long short

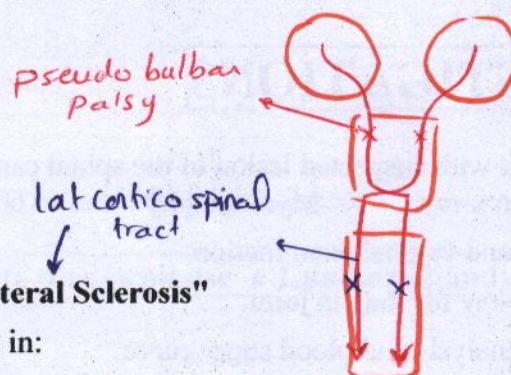
Oral sensations go!
? MND
Never can be lost

I. UMN AFFECTION

- a) In the spinal cord: Syndrome of "Lateral Sclerosis"

Bilateral manifestations of UMNL resulting in:

- Spastic paraplegia if the lesion is below the cervical region, or
- Spastic quadriplegia if the lesion is at the cervical region (C1 to C5).



- b) In the brain stem or the cerebral hemisphere: Syndrome of "Pseudo-bulbar palsy"

1. Bulbar symptoms:

- dysphagia.
- nasal regurgitation.
- dysarthria.
- hoarseness of voice.

2. Quadriplegia & signs of UMNL in the upper & lower limbs.

3. Exaggerated palatal and pharyngeal reflexes.

4. Appearance of jaw reflex (if the lesion is above the pons).

5th nucleus
pterygoids & masseter

True LMNL
nuclei

داسیجہ فریڈریکس Causes of Tonic atrophy

1 Cervical spondylosis

2 HND

93

→ fasciculations

II. LMN AFFECTATION

The disease has a tendency to affect:

1. The AHCs: in the spinal cord,
2. The cranial nerve nuclei: in the brain stem.

or



a) In the AHCs: Syndrome of "Progressive muscular atrophy"

The AHCs, mostly affected are those of the lower cervical region and to a lesser extent those of the lumbar region, resulting in weakness with signs of LMNL i.e. wasting, hypotonia, absent babinski hyporeflexia and fasciculations.

(+) due to AHCs

b) In the cranial nerve nuclei: Syndrome of "True bulbar palsy"

1. Bulbar symptoms:

- dysphagia. - nasal regurgitation.
- dysarthria. - hoarseness of voice.

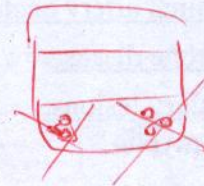
2. There is no quadriplegia, or emotional changes.

3. Absent palatal and pharyngeal reflexes.

(no) jaw reflex

~~4. There are no emotional changes.~~

→ 5. The tongue is wasted & shows fasciculations.



Pseudo-bulbar palsy	True bulbar palsy
1. UMNL	• LMNL
2. Associated with quadriplegia.	• No quadriplegia.
3. Exaggerated palatal & pharyngeal reflexes.	• Lost palatal and pharyngeal reflexes.
4. The jaw reflex may be exaggerated.	• Absent jaw reflex.
5. Tongue: no wasting or fasciculations.	• Tongue: small, flaccid & shows fasciculations.

III. COMBINED U.M.N. AND L.M.N AFFECTATION

بلا مت موجودہ ماسک Muscle Pyramidal
- Syndrome of "Amyotrophic lateral sclerosis":

"There are combined signs and symptoms of LMNL & UMNL":

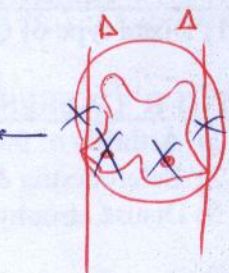
1. In the upper limbs there will be weakness, associated with wasting and fasciculations (LMNL), plus hypertonia and hyperreflexia (UMNL): This is known as TONIC ATROPHY.
2. In the lower limbs there will be weakness with signs of UMNL.

TREATMENT

1. Anabolics.
2. Massive doses of vitamin E and B.
3. Massage and mild exercise.

antioxidant

at level: Tonic atrophy
UMNL & LMNL



UMNL: below level

DD WASTING OF THE SMALL MUSCLES OF THE HAND

94

The small muscles of the hand are supplied by C₈ and Th₁ spinal segments. Wasting of these muscles may be due to:

1. AHC LESIONS:

1) Poliomyelitis:

- Age: 6 months to 2 years.
- Acute onset and regressive or stationary course.
- The wasting is asymmetrical, affecting L.L. more than U.L.
- No sensory changes.

2) Transverse myelitis: at C₈ and Th₁ segments.

3) Anterior spinal artery occlusion.

4) Motor neurone disease.

5) Intramedullary lesions:

- Syringomyelia. - Lower cervical tumours.

II. ANTERIOR ROOTS AND SPINAL NERVE LESIONS:

- 1) Cervical spondylosis.
- 2) Cervical Pott's disease.
- 3) Primary and metastatic tumours of the cervical vertebrae.
- 4) Fracture and dislocation of the cervical vertebrae.
- 5) Cervical neurofibromatosis.

III. LOWER BRACHIAL PLEXUS LESIONS:

Thoracic outlet (inlet) syndrome: could be due to:

- a) Cervical rib.
- b) Pancoast tumour:
- c) Enlarged cervical lymph nodes.
- d) Aneurysm of the subclavian artery.

IV. PERIPHERAL NERVE LESIONS:

- 1) All causes of mono, mononeuritis multiplex and polyneuropathy.
- 2) Carpal tunnel syndrome:
 - Due to compression of the median nerve in the tunnel.
 - Mainly in middle-aged women: it may be secondary to: Myxoedema, Acromegaly & Rheumatoid arthritis.
 - The thenar muscles are mainly affected, with wasting and weakness.
 - Pain and paraesthesias especially at night associated with sensory loss over the palmar surface of the fingers.

V. MUSCLE LESIONS:

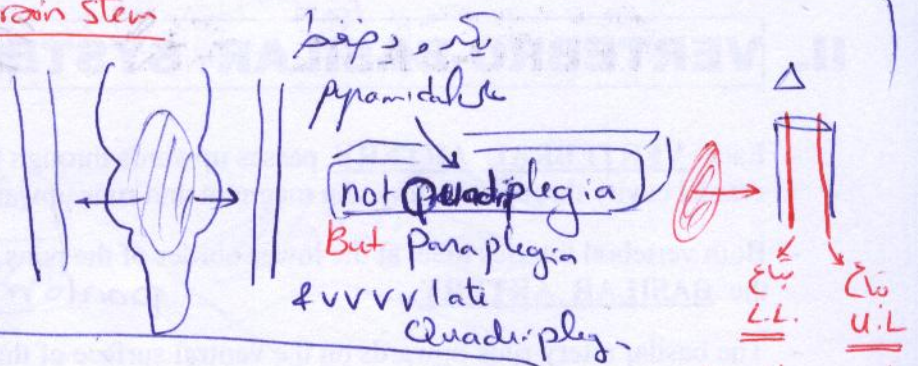
- 1) Distal type of Gower myopathy.

VI. OTHER CAUSES:

- 1) Arthritis of the joints of the hand (Rheumatoid Arthritis).
- 2) Scleroderma & Dermatomyositis.
- 3) Disuse atrophy in long standing UMNL.

نظری ۲۲
۵ درجات
جاء مرتب

NB if tumor of Brain stem



when lesion - B. stem:

فقدان السيطرة على الحركات

فقدان السيطرة على الحركات

U.L. fibers يتأثر

وعلى ما يوجد U.L. يكون ما عرفت كتي

Decerebrate rigidity:

* عدم مرونة

unknown

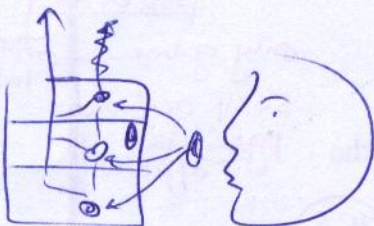
B. stem structures في

selective inhibition
extensors of U.L. & L.L.

↑↑ rigidity ← فلا يوافقوا
فقدان السيطرة

جزء
hypertonia
spastic U.L. flexors
spastic L.L. extensors

PICA



lat & medial Lemniscus
فقدان السيطرة على الحركات
lateral

فقدان السيطرة على الحركات

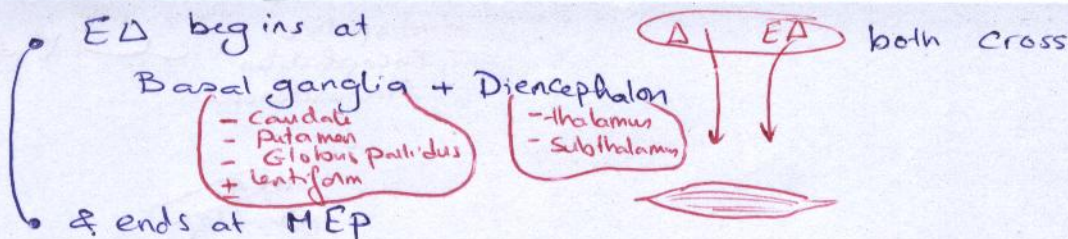
imitate facial pain

SSN

opposite side
pain & temp
(lat. lemniscus)

pain temp

→ same side



95

THE EXTRAPYRAMIDAL SYSTEM

DEFINITION

- It includes all fibres that can influence the MEP and do not pass in the pyramidal tract.

FUNCTION

1. **REGULATION** of the voluntary motor activity.
2. Regulation of the emotional & associated movements. → swinging hands on walking
3. Inhibition of the muscle tone. (not reflex) → Δ ↓ tone reflexes

DYSFUNCTION

1) **STATIC TREMORS** (disturbance in **REGULATION** of the voluntary motor activity):

- Regular: **Parkinsonism.** Both are involuntary movement
- Irregular: **Chorea, athetosis, dystonia.**

2) **BRADYKINESIA** (disturbance in **REGULATION** of the emotional & associated mov):

- Mask face (Immobile face with infrequent blinking.....staring look).
- Monotonous speech.
- Loss of swinging of the arms during walking.

3) **Rigidity (Hypertonia)** (disturbance in inhibition of the muscle tone).

PARKINSONISM (Shaking Palsy)

DEFINITION

- It is a condition in which there are **Static tremors (regular)** associated with: **Bradykinesia** & **Rigidity (hypertonia)** of the muscles of the body.

ETIOLOGY

I. **Idiopathic:**

- Age: Above 50 years.
- Sex: Both sexes are equally affected.

Deficiency of Dopamine in the Basal Ganglia

(paralysis agitans = unknown cause)

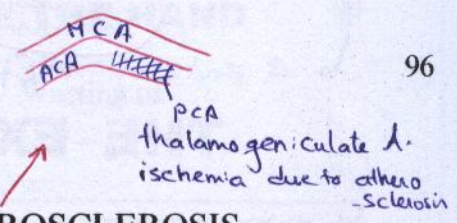
dopamine agonist

li 95

Migraine

White Knight Love

- idiopathic
- Encephalitis
- Atherosclerosis



II. Secondary:

- | | |
|------------------|--|
| 1. INFLAMMATORY: | ENCEPHALITIS. |
| 2. VASCULAR: | CEREBRAL ATHEROSCLEROSIS. |
| 3. Toxic: | Major tranquilizers (<i>Phenothiazines</i>). |
| 4. Traumatic: | Repeated trauma to the head as in boxers. |
| 5. Tumours: | of the basal ganglia. |

الور
مى
طويل

CLINICAL PICTURE

1. Static Tremors:

- Regular and occur at the rate of: 4 - 8 second.
- They give the hand the pill-rolling appearance.

as voluntary movement by Pyramidal
عقود

These tremors increase with: emotional stress & fatigue.
These tremors decrease with: sleep and during active voluntary movements.

2. Bradykinesia: Loss of emotional and associative movements

- Mask face (Immobile face with infrequent blinking.....staring look).
- Monotonous speech.
- Loss of swinging of the arms during walking.

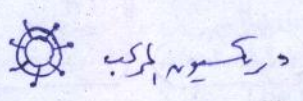
3. Rigidity: Hypertonia

A) Distribution:

- Proximal more than the distal muscles.
- Flexors of the neck, trunk & limbs resulting in the: *gorilla-like attitude*.

B) Character:

- Lead pipe: present throughout the act to the same degree, or:
- Cog wheel: interrupted by the tremors.



C) Gait:

- Short steppage: slow shuffling (Difficulty in starting the act of walking).

of V.B. Artery
Decerebrate

	Rigidity	Spasticity
Site of lesion	- Extra-pyramidal.	- Pyramidal.
Distribution	- Proximal more than distal. - Flexors of ULs - Flexors of LLs	- Distal more than proximal. - Flexors of ULs, } <i>antigravity</i> - Extensors of LLs.
Character	- Lead pipe or cog wheel.	- Clasp knife
Deep reflexes	- Normoreflexia or Hyporeflexia.	- Hyperreflexia.

Extensor UL
Extensor L.L.

⊕ MS power normal weak
⊕ Gait shuffling Circumducting
difficult although rigidity > tremors
due to severe stiffness & rigidity
may reach clonus
not smooth due to interruption by tremors
tremors > rigidity
White Knight Love

The most common causes of Parkinsonism are:

- 1) Paralysis agitans. 2) Post-encephalitic. 3) Atherosclerosis.

Features	Paralysis agitans	Post-encephalitic	Atherosclerosis
Age	Above 50 years	Any age	Above 50 years
Onset	Gradual	Usually <u>acute</u>	Gradual
Course	Progressive <i>الهزة تزداد سنياً</i>	Regressive or stationary	Remissions & exacerbations
Rigidity	Tremors more than rigidity <i>الهزة أكثر من التصلب</i>	Rigidity & tremors equally marked	Rigidity more than tremors <i>head pipe</i>
Possible associated features		Diabetes insipidus <i>itis → of hypothalamus</i>	Hypertension Coronary heart disease Diabetes mellitus <i>insulin resistance</i>

97
في بعض الحالات
الأثر
التهتك

due to
open of
collaterals

due to
atheroma
ischemia

insulin
resistance

TREATMENT

Q: Antiparkinsonial drugs

I. Medical

- The manifestations of Parkinsonism are due to an imbalance between the levels of:
↑ **acetylcholine** & ↓ **dopamine** in the basal ganglia.
- Reduction of dopamine and relative increase in acetylcholine levels are found in all cases of Parkinsonism, whatever the cause.
- Therefore medical treatment aims to restore the balance by:
 - Decreasing acetylcholine
 - or:
 - Increasing dopamine levels.

1) Anticholinergic drugs:

- a. Natural Belladonna alkaloids are rarely used nowadays. They include:
- Atropine sulphate.
 - Hyoscine.

b. Synthetic Belladonna-like alkaloids:

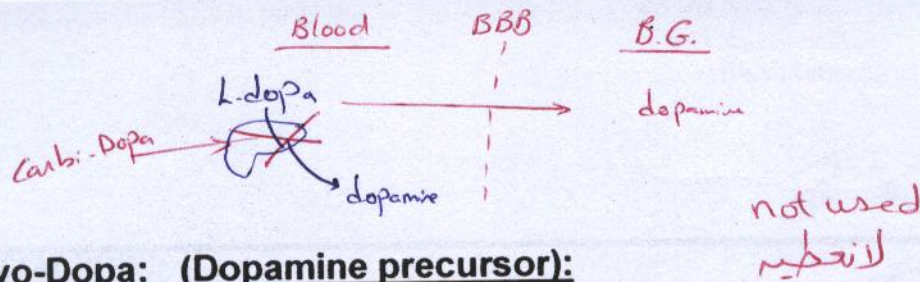
- * Cogentin 2 mg tab.
- * Artane 2 and 5 mg tab.
- * Parkinol 2 and 5 mg tab.

Dose: 1-2 tab. t.d.s., according to the severity of the case.

Side Effects: blurring of vision, dryness of mouth, retention of urine.

Toxic Effects: confusion and hallucinations.

Anticholinergics are best indicate in: TREMORS



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2) Levo-Dopa: (Dopamine precursor):

- As dopamine does not cross the BBB, its precursor levo-dopa, which can cross it, is used instead.

Dose: $\frac{1}{2}$ g orally daily; increase it by $\frac{1}{2}$ g every 3 days. Max. dose: 4-6 g daily.

Side Effects:

- 1) Psychiatric: depression, confusion.
- 2) Cardiovascular: palpitations, arrhythmias.
- 3) Gastrointestinal: nausea, vomiting.
- 4) Neurological: chorea, hypotonia, epilepsy.

3) Levo-Dopa + Carbi-Dopa: (Sinemet)

- It was found that L-dopa is to some extent decarboxylated in the liver to dopamine before crossing the BBB resulting in:

1. The appearance of side effects due to the peripheral action of dopamine.
2. The necessity of using large doses of L-dopa to insure the passage of an effective dose across the BBB.

- These disadvantages are reduced by the addition of **Carbi-dopa** which inhibits the extracerebral decarboxylation of L-dopa to dopamine.
- Carbi-dopa itself does not cross the BBB.

Dose: One tab. of Sinemet contains 250 mg L-dopa + 25 mg Carbi-dopa.

Start with one tab. daily & increase the dose by $\frac{1}{2}$ tab. every 3 days till the case improves.

Sinemet is best indicate in: BRADYKINESIA & RIGIDITY

4) Dopamine agonists:

- They mimic the action of dopamine at receptor sites:

- Trivastal:
- Bromocriptine:

20 mg t.d.s in conjunction with Sinemet.

2.5 mg daily in conjunction with Sinemet.

Bromocriptine:

1. Parkinsonism
2. Acromegaly
3. Hepatic encephalopathy

5) Amantadine hydrochloride:

- It prevents the uptake of dopamine by the neurones: 100 mg tab. t.d.s.

6) Muscle relaxants: to minimize rigidity.

II. Surgical

In severe cases

- 1) Pallidectomy. *Globus pallidus*
- 2) Thalamotomy.

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White Knight Love

CHOREA

DEFINITION

Involuntary STATIC movements of any part of the body:

- Irregular.
- Sudden & Jerky. *sudden onset*
- Pseudopurposive. *offset*

ETIOLOGY

I. Herido-familial: Huntington's chorea.

II. Secondary:

1. Autoimmune: Rheumatic chorea. *altered antigenicity*
2. Infective: Post-encephalitic chorea. *antigenic similarity*
3. Vascular: Hemiballismus. *Hge of subthalamus*
4. Toxic: Chorea gravidarum.
5. Neoplastic: Tumours of the Basal Ganglia.
6. Metabolic: Hepatolenticular degeneration (Wilson's Disease). *disturbance of ceruloplasmin*
7. Idiopathic: Senile chorea. *lenticular nucleus of B.G. liver*

$\uparrow Cu$

also
H.A.

RHEUMATIC CHOREA (Sydenham's Chorea)

- It is one of the major criteria of rheumatic fever. *وجع ازالى يتحرك فجأة*
- It may be associated with other rheumatic manifestations, but never with rheumatic arthritis.
- Age: 5-15 years.
- Sex: females more than males.

RF $\begin{matrix} \sigma \\ = \\ \phi \end{matrix}$ but chorea $\phi > \sigma$

CLINICAL PICTURE

1. Choreic movements:

a) Affect the proximal muscles more than the distal muscles:

- When the patient is asked to keep his tongue protruded and unsupported by his teeth, he is unable to do so, and quickly retracts it. *عيب في السيطرة*
- Grimacing, jerking of the shoulders, shaking of the hands and feet. *Braking*

b) They increase with emotional stress and disappear during sleep.

RF. انقلد دلوقتي في كتاب
لان كل كتاب رقة اسفله

2. Hypotonia: *as facilitatory Fibers lesion $\rightarrow$ $\uparrow$ tone*

- Choreic hand (scaphoid or boat-shaped hand):

When the patient stretches his arms there is flexion at the wrist and overextension at the metacarpophalangeal and interphalangeal joints with fanning of the fingers.

MCP PIP DIP

3. Emotional instability:

- Sudden laughter or sudden crying.

also MS

TREATMENT

1. Classic treatment of Rheumatic fever:

- Complete rest in bed.
- Corticosteroids as prednisone:
- Acetyl salicylic acid:

*1 mg / Kg / day.
6-8 gm daily.*

*on 5th week for 8 weeks,
as Carditis may be associated*

2. Specific treatment of Rheumatic chorea:

- Reserpine : 1 mg t.d.s.
- Haloperidol: 3 mg t.d.s.
- Tranquilizers.

انقل هناك دلوقة

*neuroleptic $\downarrow$ excitability of neurones
 $\downarrow$ neuronal discharge frequency*

*Reserpine
Anti HTN*

THE CEREBELLUM

The cerebellum can be divided into 3 main parts:

1. Archicerebellum:

- **Function:**
 - Maintenance of Equilibrium.
- **Lesion:**
 - Disturbance of Equilibrium.

2. Neocerebellum:

- **Function:**
 - Coordination of the voluntary motor activity.
- **Lesion:**
 - Incoordination of the voluntary motor activity.

3. Paleocerebellum:

- **Function:**
 - Maintenance of the muscle tone.
- **Lesion:**
 - Hypotonia.

PERIPHERAL NEURITIS

= neuropathy

DEFINITION

Inflammation or degeneration of the peripheral nerves and / or the cranial nerves.
Decreased conductivity of these nerves → motor, sensory & autonomic manifestations.

one or more

their roots are in sp cord

their roots are in B. stem

ANATOMICAL CLASSIFICATION

1. Mononeuropathy: affecting a single nerve in one limb.
2. Multiple Mononeuropathy: affecting 2 or more nerves in one limb.
3. Polyneuropathy: affecting many peripheral nerves in all limbs simultaneously.

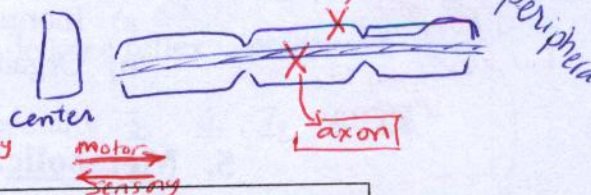
mononeuritis = multiplex

Bilat symmetrical in U.L & L.L.

PATHOLOGY

- Axonal degeneration: e.g. DM.
- Demyelination: ↓ conduction e.g. GBS.

vit. especially B12



ETIOLOGY OF MONONEUROPATHY

1. Injury: Traumatic injection into a nerve. in sciatic N.
2. Infection: Leprosy, Herpes zoster.
3. Invasion: Tumour. eg Parotid → Facial / eg CPA tumor
4. COMPRESSION: Carpal tunnel syndrome.
↳ flexor retinaculum → Median N.
= Entrapment neuropathy.

oral causes

ETIOLOGY OF POLYNEUROPATHY

I) Heridofamilial:

- 1 Hypertrophic interstitial polyneuropathy.
2. Peroneal muscle atrophy.
3. Freidreich's ataxia.

thick interstitial tissue of nerve
↓
mis- to k
↓
Conduction ↓

congenital degenerati
1- Cerebell
2- Δ
3- post col.
4- p. N.

Idiopathic

1. R. A.
2. Acromegally
3. Myxedema
4. Obesity

++ proliferat
synovium

++ GH
MPS

White Knight Love
fat

نفسه إلى تخرن

DM

- 1 Hypertrophic interstitial PN
- 2 Acromegaly
- 3 Myxedema
- 4 Leprosy

II) Secondary:

1. Infective:

- | | |
|-------------------|---|
| a) Viral: | mumps, measles. |
| b) Bacterial: | diphtheria, typhoid, tetanus. |
| c) Mycobacterial: | leprosy. Mono poly |

2. Immune:

- Collagen diseases as: RA, SLE, PAN & Scleroderma.
- Guillain – Barré syndrome. it is double etiology demyelination Begins by infection completed by Autoimmunity

3. Iatrogenic:

- INH, cycloserine, phenytoin.
= hydantoin = epanutin anticonvulsant IB anti epileptic

4. Toxic:

- | | |
|---------------|--|
| a) Inorganic: | heavy metals (lead, arsenic, mercury). |
| b) Organic: | alcohol. أكثر حاجة تعاني من الألكوليات GIT & Nerves |

5. Metabolic:

- Diabetes mellitus, renal failure. glucose metabolism

6. Endocrinal:

- Acromegaly, myxoedema.

→ ++ GH → nerve thickening

GRF PN in Myxedema

- ① deposition of MPS تخثر البروتين
- ② ↓ thyroxin → slow conduction
- ③ ↓ thyroxin → slow Intestinal absorption Jrit. B

7. Neoplastic:

- Bronchial carcinoma (paramalignant syndrome).
Small (Oat) cell ca.

UNKNOWN mechanism

8. Nutritional:

(Vitamin deficiency)

- | | |
|--------------------------------|-----------------------------------|
| • B1: Thiamine | Beri-Beri. |
| • B6: | Peripheral neuritis & dermatitis. |
| • B12: | SCD. |
| • B complex especially niacin: | Pellagra. |

B₃

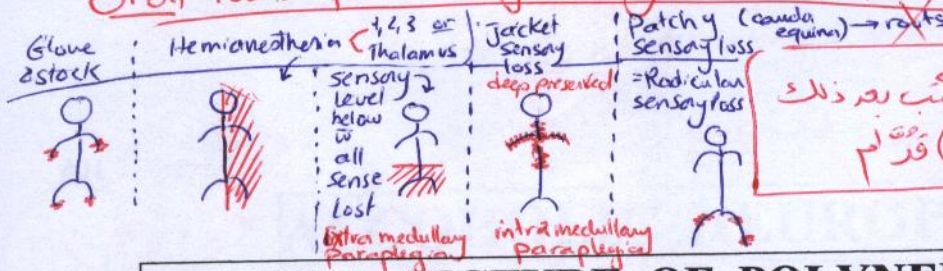
↓ by ↓ tryptophan a.a. (pts of ↑ biological value)

mainly sensory

also HDC ↑ CO failure

لأنه يتحول إلى niacin

Oral 100% Patterns of sensory loss? مكانه، لفظه، مكنه



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CLINICAL PICTURE OF POLYNEUROPATHY

- It will present by the 3 following manifestations in difference combinations.

A. Motor:

اشرح هذه الجدول مع عدم ذكر basculata

- Weakness or paralysis of LMN nature (wasting, hypotonia, hyporeflexia. ..).

- The weakness and wasting are:

- Bilateral and symmetrical.
- Affecting LL:
- Affecting distal muscles:
- Affecting extensors:

more than UL.

more than proximal muscles.

more than flexors. unknown

مشكل قادر يفقد يده ورجلاه
Bilateral foot drop and wrist drop:

due to weakness in the distal extensors.

- GAIT:

high steppage due to the foot drop.

- REFLEXES:

as distal > proximal
ankle reflex is lost, while knee reflex is preserved.

- CRANIAL NERVE AFFECTION:

especially 3, 6, 7, 9, 10.

B. Sensory:

- Symptoms:

- Pain and paraesthesia in the limbs, especially distally.

- Signs:

- Superficial sensory impairment of the stock and glove nature.
- Deep sensory loss especially distally.

at start: it is irritative
Tingle & numbness
pain, touch

late: lesion is destructive

del, i

- Muscle sense -> pinch muscle
- Joint sense -> move joint & ask him if it moves & where.
- vibration -> fork on malleolus

C. Autonomic:

- Vasomotor: coldness & cyanosis of the limbs.
- Cutaneous: loss of hair & trophic ulcers.

Ischemic manifests due to VC due to ↓ parasympathetic

NB: Regardless of the cause of the PN, the CP is essentially the same; variations depend on whether the motor, sensory or autonomic features predominate.

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INVESTIGATIONS

1. Nerve conduction velocity tests.
2. Electromyography. — to exclude that it's muscle disease

③ 3. Investigating cause
eg: DM

VALUES:

1. Confirm neuropathy.
2. Determine which nerves are involved.

SPECIFIC TYPES OF PN

CHARCOT - MARIE - TOOTH DISEASE

Mainly not only
Motor

“PERONEAL MUSCLE ATROPHY”

- **Age:** A hereditary type of PN appearing during the 1st and 2nd decades.
- **Onset & course:** Gradual onset and a very slowly progressive course.
- **Clinical picture:**

1. MOTOR MANIFESTATIONS:

not muscle disease but nerve disease
2nd 1st nerve to be affected is of peroneal MS

The wasting & weakness start in the LLs:

- In the peronii muscles then the anterior tibial group, then, resulting in:
- In the muscles of the lower 1/3 of the thigh,

“Inverted champagne - bottle appearance”

- Mild weakness despite severe wasting.

GBS: Severe weak 1st 2nd 3rd 4th 5th 6th 7th 8th 9th 10th 11th 12th wasting ↓ ↓ ↓ ↓ ↓ ↓ ↓ ↓ ↓ ↓ ↓ ↓

v. gradual onset — allows for muscle adaptation around

2. SENSATIONS:

are diminished especially vibration sense.

3. SKELETAL DEFORMITIES:

are usually present e.g. pes cavus.

Oral causes of pes cavus

= highly arched foot

- Peroneal MS atrophy
- Duchenne myopathy
- Friedreich's ataxia
- Syngomyelia

ازای طرف آن PN سببی شرب الگوهای
H/o prolonged intake

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ALCOHOLIC NEUROPATHY

1. Mainly sensory.

2. Associated with Wernick's encephalopathy: *Amnesia* + *Ataxia* + *Ophthalmoplegia*.
toxic effect

LEAD NEUROPATHY

1. Purely motor.

2. Associated with other features of chronic lead poisoning:

- Anemia: Hemolytic anemia, Sideroblastic anemia.
- Hypertension & lead encephalopathy.
- Blue lines in the gums.

ARSENIC NEUROPATHY

1. Mainly sensory.

2. Associated with other features of chronic arsenic poisoning:

- Hyperkeratosis of the skin.
- Depigmentation of the skin.

DIABETIC NEUROPATHY

Diabetic neuropathy is the most common complication of diabetes, both types 1 & 2.

Pathogenesis:

METABOLIC FAILURE

- Hyperglycemia: → harmful biological changes → nerve damage.
- Ketone bodies: DKA → toxic to the nerves → nerve damage.
- Polyuria: → hypovitaminosis (B₁, B₆ & B₁₂) → nerve degeneration.

VASCULAR INSUFFICIENCY

- Diabetic microangiopathy: of the vasa nervosa.
- Ischaemia of the nerves: due to atherosclerosis of the vasa nervosa.

Aceto acetic acid

Hydroxy
Butyric
acid

Small
Bv.

PN
neuropathy
retinopathy

Large
Bv.

3rd
Common
↑ LDL

2nd
Common
↓ HDL

Coronary
Cerebral
vasa nervosa

dyslipidemia

& the most common is
↑ Triglycerides
White Knight Love

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Clinical picture:

1. SENSORY MANIFESTATIONS:

- It is **mainly sensory**.
- It starts as mononeuropathy & later becomes polyneuropathy.
- **Superficial sensory affection:**
 - It starts with pain and paraesthesia followed by stock and glove hyposthesia.
- **Deep sensory affection:**
 - It starts with ↑ muscle sense → tender calf followed by lost muscle sense.
 - There is also **loss of deep reflexes and sensory ataxia**.

2. MOTOR MANIFESTATIONS:

- Minimal.
- Late.

3. AUTONOMIC MANIFESTATIONS:

- **Impotence.**
- **Syncope (postural).**
- **Incontinence.**
- **Silent myocardial infarction.**
- **Trophic ulcers.**

Investigation: 1 Nerve conduction study
2 Blood Glucose

Treatment:

1. **Proper control of diabetes.** eg Insulin or oral drugs (S) (II)
2. **Pain control:**
 - Analgesics.
 - Anti-seizure medications: Gabapentin, Carbamazepine.
3. **Physiotherapy:** in case of motor weakness.
4. **Vitamins B complex.**
5. **Vasodilators.**

++ GABA inhibitory neurotransmitter.

++ pain threshold
[↓ conduction of nerve

هو اشارة على
ان هناك مشكلة في التوصيل

DIPHThERITIC NEUROPATHY

M mainly

Neuritis is the commonest and most important nervous complication of diphtheria.

Etiology:

- Nerve damage by the exotoxin of *Corynebacterium diphtheria*.

Clinical Picture:

- It is **mainly motor**.
- Symptoms and signs occur 2-8 weeks after the appearance of diphtheria.
- Two main types of neuropathy may occur:
 1. *Localised type:* affecting the Cranial nerves: 3, 7, 10. *only*
 2. *Generalised type:* affecting the Peripheral nerves.

*أسهل
لـ
الـ*

LEPROTIC NEUROPATHY

*أسهل
S mainly*

Etiology:

Causative organism: *Mycobacterium leprae*.

Nerve infiltration by leprous granuloma → thickening, degeneration & ↓ conductivity.

*Chronic
inflamm.*

Clinical Picture:

- The neuropathy is mainly sensory.
- The neuropathy may be of the mono-or polyneuritic types.
- Commonest nerves affected are: lateral popliteal, ulnar, greater auricular, trigeminal & facial.

*Ulnar N.
مصابة
بـ
الـ*

Clinical Types:

1. Nodular (lepromatous) leprosy:

- Cutaneous nodules over the face & neck which ulcerate & heal by fibrosis → **leonine face**.
- Associated with fever and lymphadenopathy.

RES hyperplasia

2. Maculo-anaesthetic (tuberculoid) leprosy:

- Patchy depigmented areas.
- Patchy sensory loss.

*أسهل
لـ*

3. Mixed type.

أسهل

Treatment:

- Dapsone: 100 mg / day, orally for 2 years.
- Rifampicin: 600 mg / day, orally for 6 months.

or

*except if Resistant
→ use both*

*Oral
Anti-leprae
Anti-TB
Anti-Biotic*

*Prophylactic Curative
Brucella
White Knight Love*

[All vit B ↓ → mainly sensory
Most vit B ↓ → Demyelination

SQ
vit B deficiency 110

BERI - BERI

Etiology:

Vitamin B1 (Thiamin) deficiency.

Clinical Picture:

1. Dry beri-beri:
2. Wet beri-beri:
3. Wernicke's encephalopathy:

peripheral sensory neuropathy.

picture of congestive heart failure.

Amnesia, + Ataxia + ophthalmoplegia.

TREATMENT:

- Thiamine 100 mg daily and vit. B complex.
- Treatment of Heart failure.

PELLAGRA

Etiology:

Multiple deficiency disease due to vitamin B complex deficiency especially niacin; due to:

1. Decreased intake of vitamin B complex.
2. Decreased intake of proteins of HBV (containing nicotinic acid or its precursor tryptophan).
3. Decreased absorption as in gastro-enteritis.
4. Parasitic infestation.

Clinical Picture:

I. Cutaneous manifestations:

- Bilateral and symmetrical dermatitis followed by hyperkeratosis.
- The lesions involve the exposed areas, and over bony prominences esp. Trochanters

II. Gastro-intestinal manifestations:

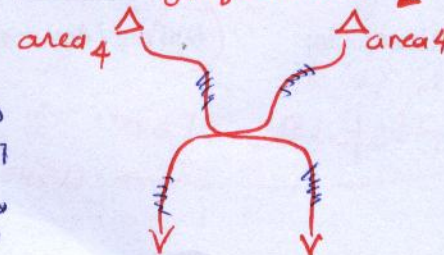
- Mouth: stomatitis, glossitis with atrophy of the papillae (glazed tongue).
- Stomach: dyspepsia, nausea, vomiting, epigastric pain.
- Intestines: diarrhoea.

III. Neuro-psychiatric manifestations:

- PN mainly sensory due to:
- Para - or quadriplegia due to:
- Mentality changes as:

PN degeneration.
Pyramidal tract degeneration (pellagral lateral sclerosis).

dementia.



White Knight Love

Treatment:

1. Treatment of the cause. *eg parasitic infestation*
2. Nicotinamide 500 mg IV / day.
3. Vitamin B complex. *absorption*

SUBACUTE COMBINED DEGENERATION*B₁₂***SCD****Etiology:**Vitamin B₁₂ (cyanocobalamin) deficiency:

"Refer to Hematology".

Clinical Picture: Triad of: *Anemia + GIT + Neurological manifestations.***I. Anaemia:**

(megaloblastic, refer to BLOOD).

*اندرج کالی
رکتی
Blood
megaloblastic***II. Gastrointestinal manifestations:**

- Refer to BLOOD.

*اندرج کالی
رکتی
Blood**Megaloblastic***III. Neurological manifestations:**- Combined degeneration of the Peripheral nerves, Posterior columns & Pyramidal tracts.**1. Superficial sensory affection:**

Pains and paraesthesias in the limbs followed by stock and glove hyposthesia.

*Coordinations
Balance***2. Deep sensory loss:**Sensory ataxia due to Posterior column degeneration.Ankle reflex is lost while knee reflex is preserved due to Peripheral nerve degeneration.*Proximal < Distal
(but exaggerated due to Δ affection)***3. Limb weakness with + ve Babinski, BUT: with hypotonia & hyporeflexia:**Weakness in the limbs with + ve Babinski: due to Pyramidal tract degeneration *UMNL*

Hypotonia & Hyporeflexia:

due to Peripheral nerve degeneration. *LMNL**[Distally]
ankle only
not knee**quadri or Para
Plegia (never hemiplegia)***Treatment:**

- Vit B₁₂ (1000 µgm IM daily for 2 weeks then 100 µgm twice per week).

*اندرج کالی
رکتی
Blood*

ACUTE INFLAMMATORY DEMYELINATING POLY - RADICULO - NEUROPATHY

AIDP

Guillain-Barré Syndrome

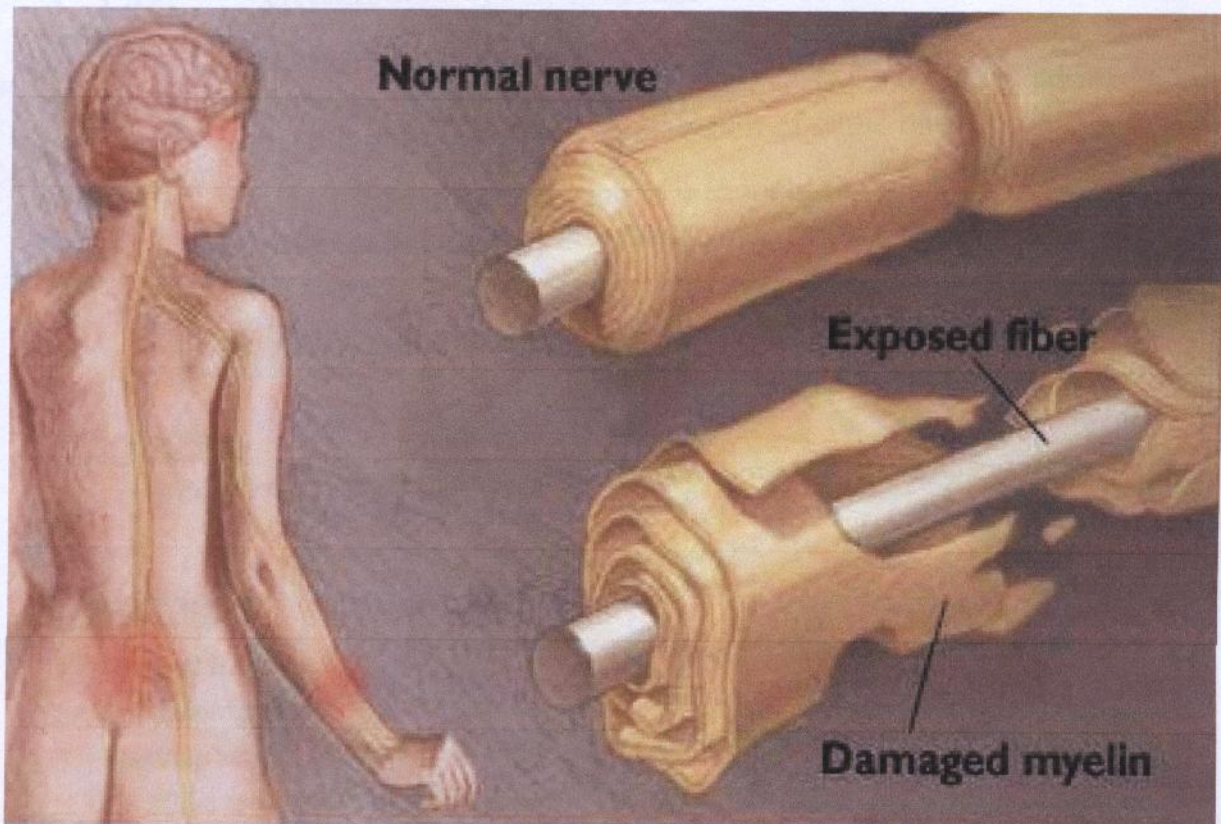
Etiology: "Double etio"

- It is due to an **auto-immune** disease following upper respiratory or GIT **infection**.
- Infection → antibodies against the myelin sheath of the nerves → Demyelination.

Infecting organisms

- Bacteria: *Campylobacter jejuni* (most common) (Gram - ve bacteria).
- Viruses: Cytomegalovirus (second most common), EBV.

Nerve damage in GBS (Demyelination)



* also other disease
same pathway < infects
end effect

eg: AIDS
العدوى
تنتج 10

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Clinical Picture:

Infective

1. Febrile stage: Influenza-like attack with:

- Fever, headache, malaise, pains all over the body, with no nervous symptoms.

ما يحدث يحصل خلل
في المناعة
(Abs)

2. Latent stage: Symptoms disappear: the patient is free for 1-3 weeks.

الفترة التي فيها Abs يتطالع

3. Paralytic stage: as mainly Motor

Neuro-
logical

myoathenra
is descending

also
myoathenra

- Weakness with an **ascending march**: There is acute severe weakness or paralysis starting in the: LL muscles and ascending to involve the trunk and respiratory muscles, followed by the UL muscles.
- Weakness **proximal more than distal**: contrary to other types of PN.
- **No wasting**: In spite of the severe degree of paralysis. as acute onset with v short duration
- **Sensory impairment**: Stock and glove hyposthesia and deep sensory loss.
- **Cranial nerve involvement**: especially 7, 9, 10.

Acute
Restrictive
Resp.
failure
type II
as hypo-
ventilatory
hypoxia hyper-
capnia
طيف
type I
نقص
الانتشار

Investigations:

Cells cyto ↑ mild
L. ptn alb ↑↑↑

1. CSF: "Cytoalbuminous dissociation" (↑ proteins more than the cells).

- During the acute phase of GBS, the characteristic findings include cyto-albuminous dissociation, which is an elevation in CSF protein without an elevation of white blood cells.
- The increase in CSF protein is thought to reflect the widespread inflammatory disease of the nerve roots.

Stroke
Bloody clindensation

① ② Abs ↑↑

2. Serology:

- Antibodies to glycolipids of the myelin sheath: +ve in 70 % of patients.

IgG Abs
ومفوض مرضه من غير GBS يطالع هذا النوع من Abs

sensitive
specific

Prognosis:

- Prognosis is good in 85% of the cases where recovery occurs in 3-4 weeks.

حتى غير علاج
ولو خفف يمتد
Acute Resp. failure

Treatment:

General

1. General lines:

bed rest, vitamins, physiotherapy.

2. Corticosteroids:

Prednisone, 1 mg / Kg / day.

3. Plasmapheresis (plasma exchange):

the treatment of choice.

"The mechanism of plasmapheresis is the removal of antibodies from the serum".

4. IV Gamma-globulins:

as effective as plasmapheresis, they inhibit antibody production.

5. TTT of respiratory failure: " refer to CHEST.

Extracorporeal
أخرج دم برة
جسمه بأخوش
منه Abs وأدخل
ناتج

أخوش
Abs

by
Phoresis

113

Cortisol
or

IV Gamma
globulins

Abs
White Knight Love

انسع
Resp failure
كل

3 diseases & cytoalb. dissociate

1. GBS
 2. Extra Medullary paraplegia
 3. Multiple sclerosis (MS)
- ptn ↑↑↑ , cells ↑
ptn ↑ , cells ↑↑↑

Oral 1000
لا يتبع 8 في
المرحلة
لا تتركه الا لو شل عنده

plasma pheresis indications

* IgG ✓✓

(IgM كلها)
جميع كبر لا تتخل
في المحرار الك
خروج في الفلتر

من اي عيان
Auto immune
ما تتخل
plasma pheresis
لا على حسب
نوع Ab

CAUSES OF MOTOR NEUROPATHY

1. Peroneal muscle atrophy.
2. Lead neuropathy.
3. Diphtheritic neuropathy.
4. GBS.

CAUSES OF SENSORY NEUROPATHY

1. Alcoholic neuropathy.
2. Arsenic neuropathy.
3. Diabetic neuropathy.
4. Leprotic neuropathy.
5. Vitamin deficiency neuropathy.

CAUSES OF LOST ANKLE REFLEX WITH PRESERVED KNEE REFLEX

- | | |
|--|--|
| 1. Epiconus lesion. | 4. Subacute combined degeneration (SCD). |
| 2. Cauda equina lesion affectin S1 root. | 5. Friedreich's ataxia. |
| 3. Peripheral neuropathy. | |

+
 lost ankle
 exaggerated knee
 as both
 SCD
 Friedreich's
 = Δ tract affection

2ry
Paramalignant
syndrome
منه غير متوافق

Myasthenias

1ry

Clinical
DD

من نظري
ولكن شفوي

نظر
البيوتو

MYASTHENIA GRAVIS

DEFINITION

- It is a disorder of transmission at the **NMJ** characterized by:

weakness and fatigability of skeletal muscles.
impulse
on repetition of movement

ETIOLOGY

Auto-immune: may be associated with autoimmune diseases (RA, SLE)

1. Acetylcholine Receptor (AChR) Antibodies:

- There is production of antibodies against the AChR of the **NMJ** leading to their destruction.

2. Thymic Dysfunction: e.g. Thymic hyperplasia or Thymoma

- Thymic dysfunction may lead to disturbed immune response with production of AChR antibodies.

CLINICAL PICTURE

"Females 20 - 40"

- Onset and Course: usually gradual onset and progressive course.
- Muscles: easy **fatigability** on **repetition** of movement.

- The disease affects **ONLY** the skeletal muscles in a **DESCENDING** manner:

1. **The ocular muscles** leading to: **ptosis**, **diplopia** & **ophthalmoplegia**.
2. **The jaw muscles** leading to: the mouth hanging open.
3. **The facial muscles** leading to: an expressionless appearance. **Mask face**
4. **The bulbar muscles** leading to: tetrad of bulbar symptoms.
 - dysphagia
 - dysarthria
 - nasal regurgitation
5. **The skeletal muscles of the body:**
 - Muscles of the UL are affected before those of the LL.
 - Proximal muscles are affected more than the distal muscles.
 - Respiratory muscles are affected leading to dyspnea & respiratory failure.

- **Diurnal variation** is noted: motor power is better in early morning than at **end** of day.

- Myasthenic crisis:

- It is characterized by aggravation of the condition & severe respiratory distress.
- It is precipitated by: **infections**, **operations**, **OR** **without a cause**.

INVESTIGATIONS

1. **Serology:** AChR antibodies are detected in the serum of 90% of patients.
2. **EMG:** Repetitive nerve stimulation (RNS) → decremental response in action potentials.
3. **Imaging:** MRI or CT of anterior mediastinum: may show **Thymus enlargement**.
4. **Prostigmine therapeutic test:** IM Prostigmine causes improvement of fatigue after **20 min**.
5. **Muscle biopsy:** Increased lymphocytes.

Anti **Acetylcholine** esterase

ACh is produced
→ no response
So, another amount
produced till depleted
from presynaptic
stores → fatigue
on repeating
movement

* **Bilat ptosis**
• M. Gravis
• Congenital
* **unilat ptosis**
• Horner
• III Cranial

Oral → **Mask face**
1. LMNL facial
2. M. Gravis
3. Parkinsonism

4. Scleroderma
5. Myxedema

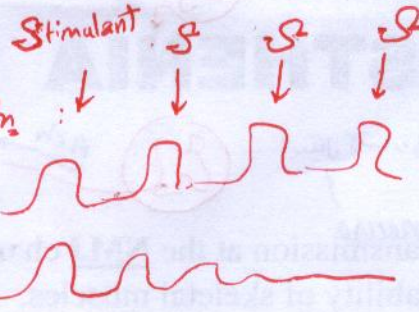
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★ EMG

if Repeated stimulation

• normally

• M. Gravis



↳ & stimulation is for nerve

ولا لو علت لا Ms



لا فارغ من M. Gravis و الـ

★ $\uparrow K^+$

SK. Muscle

Cardiac Muscle

(arrest in diastole)

$\downarrow K^+$

SK. Muscle

Cardiac Muscle

Smooth Muscle

Urinary bladder
intestine

Resp failure by

• GBS → Acute

• M. Gravis → Acute
Chronic

Myopathy Chronic

TREATMENT

1) Medical:

1. Anticholinesterase drugs: Prostigmine: 15 mg tds orally
2. Corticosteroids: in patients not responding to anticholinesterase drugs.
3. Immunosuppressive drugs: e.g. Azathioprine or cyclophosphamide.
- These are used in patients not responding to corticosteroids.

2) Plasmapheresis: reduces the AChR antibody levels.

3) Irradiation or Surgical removal of the Thymus.

4) Treatment of Myasthenic crisis:

- IM Prostigmine, Plasmapheresis, Mechanical ventilation.

For Acute Resp. failure → death

MYOPATHIES

DEFINITION

- A disease of the skeletal muscles characterised by gradual progressive DEGENERATION.

ETIOLOGY

I. Primary Myopathy: "Progressive Muscular Dystrophy" "Hereditary" PMS

II. Secondary Myopathy: "Secondary to an underlying disease"

1. Immune: Polymyositis & Dermatomyositis. (Abs against Muscles) + (Abs against skin)
2. Iatrogenic: Alcohol, Corticosteroids, Statins, Zidovudine.
3. Endocrinal: Acromegaly, Cushing's syndrome, Myxoedema.
4. Neoplastic: Bronchogenic carcinoma (paramalignant syndrome).
5. Metabolic: ↑ K or ↓ K.

CLINICAL PICTURE

- Onset and Course: usually gradual onset and progressive course.
- Muscles: proximal muscles are affected more than the distal muscles.

INVESTIGATIONS

1. Estimation of serum enzymes: LDH, ↑↑ CPK, Aldolase, SGOT.
2. EMG: Diagnostic. Low voltage Action potential
3. Muscle biopsy: Degeneration of muscle fibres and its replacement by fibro-fatty tissue.

TREATMENT

no specific ttt, only supportive: "Vitamins, Physiotherapy".

Causes of death in Myopathies

Respiratory failure, Orthostatic pneumonia, DCM (in Duchenne type)

positional
بسبب الرقبة لأنه شل

H.F.

White Knight Love

BRAIN TUMOURS

CLASSIFICATION

I. Primary Tumours:

- From the meninges: Meningiomas.
- From the brain tissue: Gliomas.
- From the pituitary: Pituitary tumours.
- From the cranial nerves: Acoustic neuroma (of the 8th nerve).
- From the blood vessels: Hemangiomas & Hemangioblastomas.

II. Secondary Tumours:

- Metastasis.

but esp. ← Breast, prostate, Bronchi

CLINICAL PICTURE

- Manifestations of Increased ICT.
- True localising manifestations.
- False localizing manifestations.

- space occupying lesion causing ↑ ICT
- obstruct
- damage of BBB → Brain edema
- Neovascularization
- ± hge = Brain

1. Manifestations of Increased ICT

1. HEADACHE

- Dull aching.
- Severe in the **morning** & then tends to diminish.
- Increases by: coughing, sneezing & straining.
- It has no significant value in localization of the site of the tumour.

Due to stretch of the meninges

Due to CSF congestion as venous circulation is stagnant in sleep

Pain sensitive

Normally pressure: = 15 mm.Hg

if > 20 = ↑ ICT

as sympathetic + venous drainage → CSF drain

venous return

2. VOMITING

- Projectile: not related to meals & not preceded by nausea.
- More frequent in the **morning**.

Due to stimulation of the vomiting centre

CSF congestion as venous drain is minimal

Part of generalized edema of brain

Optic N. surrounded by meninges & B.V.

closed vein & open Artery

المسحوق ولا يفتح

3. PAPILLOEDEMA

A reliable sign of increased ICT as

- Blurring of vision occurs followed by gradual diminution of vision.
- Failure of vision occurs due to post-papilloedemic optic atrophy.
- Ophthalmoscopically there will be:

- Blurring of the margins of the optic disc.
- Retinal hemorrhages & exudates.
- Optic atrophy. (irreversible)

after vomiting → ↓ Body fluid → partial rel. of

2. True Localizing Manifestations

- These are **specific** symptoms & signs that depend on the **specific** site of the tumour.

1. FRONTAL LOBE TUMOURS.
2. PARIETAL LOBE TUMOURS.
3. TEMPORAL LOBE TUMOURS.
4. OCCIPITAL LOBE TUMOURS.

Refer to: areas of the Cerebral cortex

5. PITUITARY TUMOURS:

A. Suprasellar Tumours:

- ① Hypothalamic syndrome:
- ② Panhypopituitarism.
- ③ Bitemporal hemianopia.

Craniopharyngioma
Diabetes insipidus, obesity & hypersomnia.

Hypothalamus +++ Pit → ADH
D.I. (Diabetes Insipidus)

B. Intrasellar Tumours:

1. Hormonal manifestations:

- Chromophobe adenoma: Panhypopituitarism.
- Acidophil adenoma: Gigantism or Acromegaly.
- Basophil adenoma: Cushing's syndrome.

2. Headache: it passes through 3 stages

- It starts bitemporal due to increased intrasellar pressure.
- It then disappears due to rupture of the sella turcica. → decompression
- It reappears later to produce generalized headache of ↑ ICT.

3. Neurological manifestations: due to compression of adjacent structures

I. Anterior compression:

- Compression of the optic chiasma: Bitemporal hemianopia.
- Compression of the optic nerve: Optic atrophy. Ipsilateral Blindness
- Compression of the olfactory tract: Anosmia.

II. Posterior compression:

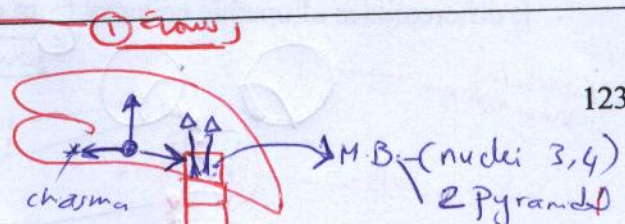
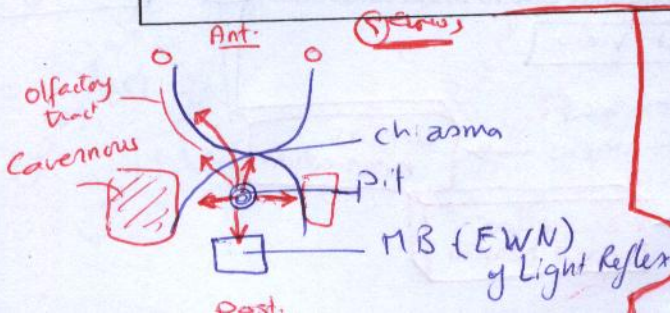
- Compression of the upper brain stem: Bilateral pyramidal signs. Ophthalmoplegia.

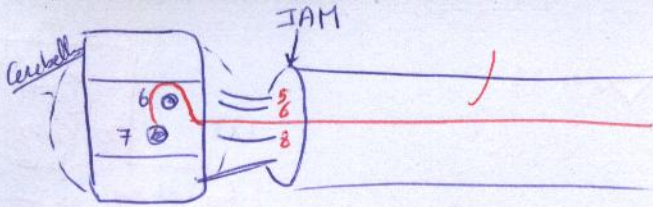
III. Lateral compression:

- Compression of the optic tract: Homonymous hemianopia.
- Compression of cavernous sinus: Paralysis of 3rd, 4th & 6th cranial nerves. Affection of ophthalmic division of 5th cranial nerve.

IV. Superior compression:

- Compression of the hypothalamus: Hypothalamic syndrome.





6. CEREBELLO-PONTINE ANGLE TUMOURS:

- Ipsilateral ataxia.
- Ipsilateral affection of: 5th, 7th, 6th, 8th cranial nerves.
- Contralateral hemiplegia (pyramidal compression in the pons).

اسمى كامل
مريض
p37

لذلك وحالة لما اسمى افكر انه الورم طالع حركته فيه انه غلط

3. False Localizing Manifestations

- These are nonspecific symptoms & signs that occur regardless of the specific site of the tumour.

1. Dilatation of the lateral ventricle:

- Mental confusion. prefrontal area affection

2. Dilatation of the 3rd ventricle:

- Hypothalamic syndrome (compression of the hypothalamus).
- Panhypopituitarism (compression of the pituitary gland).
- Bitemporal hemianopia (compression of the optic chiasma).



بالخيط مع
supra
- sellar
tumor

عنه افكره
true

3. Dilatation of the 4th ventricle:

- Disturbance in HR & BP (irritation of the vasomotor centre).
- Disturbance in respiration (irritation of the respiratory centre).
- Vomiting (RF) (irritation of the vomiting centre).

"irritation of centers"

Cardiac Inhibitory Centre

depression

failure
= embarrassment

4. Cranial nerve paralysis:

- The most commonly affected nerve is the 6th nerve as it is thin & runs a long course.

Convergent squint

5. Herniation syndromes:

100% oral

هناك
hernia
بفتح الهمزة

من طرف الورم الى زقاقه يكون
لازمه فيه

a) Herniation of the uncus of the Temporal lobe:

- Compression of the reticular formation in the MB:
- Compression of the 3rd nerve nucleus in the MB:

impaired consciousness.
dilated fixed pupil.

b) Herniation of the cerebellar tonsils:

- Compression of the medulla:

disturbance in respiration.

VMC → Hypertension
CTC → brady cardia
RC → RF

Cushing's Triad

INVESTIGATIONS

1. MRI & CT scan:

- The best & most accurate investigations.

2. EEG:

- It differentiates idiopathic epilepsy from epilepsy due to brain tumours.

Brain edema
frontal lobe irritate

Cerebral hemisphere

Cerebellum

Cerebral hemisphere
Cerebellum
& Brain stem

Tentorium Cerebelli

Tentorial notch = hiatus

2) out view

Temporal uncus

Uncus herniation

Cerebellar tonsil hernia

1) Lat view

3. Plain X-ray of the skull:

a) Signs of increased ICT:

3S

- **S**ilver-beaten appearance: *finger prints appearance.*
- **S**eparation of the cranial sutures. *if in child*
- **S**ellar changes (in pituitary tumours): *enlargement of the sella turcica.*

b) Signs denoting the site of the tumour:

- Localised calcification.
- Localised erosion.



as ↑ICT → causes that sulci & gyri Press on inner surface of skull
= Pressure markings
= Convolution "

= widening
= ballooning
= saucerization (تسطيح, كسر)



4. Cerebral angiography:

- It may show abnormal vessels feeding the tumour.

5. ^{cor} Air or myodil ventriculography:

it may show:

- Filling defect.
- Displacement of the ventricular system.

6. Ophthalmoscopic examination:

- It may show papilloedema.

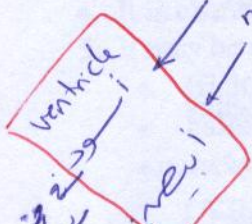


vessels
= neovascularization
= Angiogenesis
occurs in tumor



obsolete
اختارت 1918

filling defect



ventricle
تجويف المخ
البنية المخية

تسأل في قنطرة
لو حققت
" air myodil
تسأل في قنطرة
في البنية المخية
filling defect
تسأل في قنطرة
في البنية المخية
تسأل في قنطرة
في البنية المخية

Epilepsy

neurotransmitters

excitatory

Inhibitory

balance

هنا فيه خلل في هذا الميزان لصالح ان
excitatory ↑ ↓ inhibitory ↓

Excitation of Brain cells

كهرباء زيادة بالبدي

epileptogenic focus = hyper excitable neurones

تستجيب زائدة عنه للزفرة

motor actions ↑

motor area 4

وال C/P على حسب مكانه فيه

Parasthesia

sensory area 1, 2, 3

Jacksonian fit may be sensory or motor

الخطبة في
كهرباء المخ

أي ما يقرب
فيه كهرباء ولما
تلتخط بعمل
anhythmia
يصرف النظر عنه
سبب الخطبة فيه

* مريض نوع *arrhythmias* خالٍ من تشخيص بحاجة غير ECG
عالمش وجود

* وكذلك مريض نوع *epilepsy* خالٍ من تشخيص بحاجة غير EEG

الشراة باعة
epilepsy

oral

EEG is the most important investigation في النوع والأخيرة

introduction about functions of brain

Normally

Focus ← مركزية جزء من المخ أدنى المخ
سبب السبب بقاء

if disturbed "hyperexcitable"

1. Somatic functions (Bilaterally) من الجانبين

Centers in Cortex

1. Motor (area 4)

Motor Jacksonian fits

2. sensory (area 1,2,3)

Sensory

burning numbness tingling

3. special senses

olfaction (uncus)

olfactory

hallucination

vision (area 17)

visual

hallucination

auditory (41,42)

auditory

hallucination

2. Autonomic functions

Centers in Hypothalamus

1. Motor autonomic

eg: GIT motility

Bladder

heart

Abdominal colic, diarrhea, vomiting

Urine incontinence

Tachycardia

Skin { sympathetic parasympathetic }

Sweating

2. Sensory autonomic

حساسية

nausea, abdominal distension

dyspnea, palpitation

3. Psychological functions (behaviour)

Behavioural changes

Centers

1. Prefrontal area

2. Limbic system

Hallucination

تصريفات
مفكوسة

Temporal lobe epilepsy

temporal lobe epilepsy

4. Consciousness Level

Centers in 1. RAS 2. cortex

حضر لامتحة oral من أنواع
epilepsy →

White Knight Love

* aura: precedes the epilepsy:

سبب رضة ← electric discharge
كثرة وتيرة وقت قليلة
وهي من جنس ال attack

Mechanism of epilepsy → 2ry causes (General causes)

one or more of following mechanisms

دافع

1- Cortical irritation & stimulation of Brain cells and/or facilitation of neurotransmission

لا يكتب في النظرى
لكنه عشان لازم

through inhibition of inhibitory transmitters (GABA)

- eg: Fever
- eg: Electrolytes

لذلك العلاج هو دواء
يهدئ الخلايا ويزود GABA

2- Brain hypoxia

- eg: Fallots Tetralogy: infundibulum → Cyanotic spells
- eg: Adam stroke's attack: Complete Heart Block

الدم يقل
لا يدخل

Focus [idioventricular rhythm] →

لكنه ال focus
ده وقف
مفشي

3- Toxic effect "Toxins pass BBB from Blood to brain"

- eg: Renal failure
- eg: Liver failure
- eg: Alcohol

MSOF → (واحد منهم هو الخ)

ضغط نبض
مانع على ترويض
عنه تشنجات

4- Nutritional deficiency → electrical instability

- eg: ↓ B₁ (vitamins)

EPILEPSY

1 DEFINITION

- It comes from the GREEK name "**Epilepsia**" which means "**taking hold of** or **seizing**".
- It is a disorder characterized by:
 - 1 spontaneous tendency for recurrent **seizures**.
 - 2 free between attacks

SEIZURES

- Recurrent transient attacks of:** **somatic**, **psychic**, or, **autonomic** clinical features.
- Represent:** (reflects) clinical features of abnormally hyperexcitable cortical neurons.
- Result from:** **paroxysmal** and **excessive** electrical neuronal discharges.
- ASSOCIATED WITH:** EEG changes & may be disturbance of consciousness.

2 ETIOLOGY

1. Idiopathic epilepsy

- It is the commonest cause.
- It may be associated with **positive family history** in some cases.
- It starts in the 1st & 2nd decades in the form of:
 - Grand mal epilepsy.
 - Petit mal epilepsy.
 - Myoclonic epilepsy.
 - Atonic seizures.

no cause can be detected (65%) → **HCCQ**

in 30% **HCCQ**

2. Secondary epilepsy

A. Local causes in the brain:

1. Congenital: cerebral palsy.
2. Traumatic: cerebral contusion or laceration.
3. Inflammatory: encephalitis, meningitis, brain abscess.
4. Neoplastic: brain tumours.
5. Degenerative: presenile dementia.
6. Vascular: stroke (especially hemorrhagic), hypertensive encephalopathy.

B. General causes with secondary effects on the brain:

1. Toxic: Alcohol, cocaine, lead.
2. Iatrogenic: Lidocaine, INH.
3. Metabolic: ↑ glucose & ↓ glucose.
4. Endocrinal: Hypoparathyroidism → ↓ Ca.
5. Organ failure: Hepatic failure.
6. Heart disease: Adam's Stoke's attacks.
7. Nutritional: Pellagra, B₃.
8. Physical: High fevers.
9. HYSTERICAL.

- Botulism, tetanus, CO, cyanide.
- Ambilhar, Amphetamine, Aminophylline.
- ↑ Ca & ↓ Ca.
- Hyperthyroid crisis.
- Renal failure.
- Fallot's tetralogy.
- Vitamin B₆ deficiency.
- Heat stroke.

Toxic effect

Cerebral irritation effect

hypoxic effect

White Knight Love

3

CLINICAL PICTURE

1. GENERALISED SEIZURES

"Excessive electrical discharges from cortical neurons in **BOTH** hemispheres simultaneously"

I. Grand Mal Epilepsy:

"attacks of tonic-clonic convulsions"

1. Pre-ictal stage (seconds) (aura)

- It is a warning sign of a coming attack.
- It may be: (brief electrical discharge)

- Somatic:** Myoclonus, Parasthesias. (burning tingling numbness)
- Psychic:** Hallucinations.
- Autonomic:** Tachycardia, Sweating, Colic

2. Ictal stage (seizure)

- Sudden loss of consciousness: for seconds to minutes.
- Tonic phase (few seconds) = sustained contraction

- The UL & LL: are extended.
- The HEAD: is retracted to one side & the eye balls rolled up.
- The BACK: High arched (opisthotonus)
- The JAWS: are firmly clenched, with biting of the TONGUE.
- CYANOSIS: due to impaired respiration.
- There may be incontinence of urine.

- Clonic phase (few minutes) = Contraction then Relax
- The UL & LL: contract & relax repeatedly & rapidly.
- The HEAD: jerks forcibly.
- may be incontinence of urine

3. Post-ictal stage (sequelae)

- It may be:

- Somatic:** Todd's paralysis (< 24 hours, due to neuronal exhaustion).
- Psychic:** Confusion.
- Autonomic:** Vomiting.

Drug of choice: Carbamazepine (Tegretol) or Phenytoin (Epanutin)

II. Petit Mal Epilepsy: "attacks of loss of consciousness" "Absence"

- It starts in childhood & improves at puberty & usually disappears at the age of 20.
- It is NOT PRECEDED by aura & NOT FOLLOWED by sequelae.
- It is usually PRECIPITATED by: hyperventilation or photic stimulation.
- It is characterized by: sudden loss of consciousness of short duration (few seconds).
- It may be associated with:

- High frequency (50 attacks / day) = **picnolepsy**
- Falling to the ground without warning.
- Jerky movements of the head & UL (myoclonic petit mal).

Drug of choice: Valproate (Depakine) or Succinimide (Zarontin)

127

General causes of 2ry idiopathic

لو اسباب غير معروفة
status epilepticus
عقبات عت

jerky movement
contractions followed by Relaxation

Never exceed 30 min
if exceeded => status epilepticus

لذلك لا زل
فم
قطة جوة بقاء
عشان ميعطش بالسانه
R.F. تحوت

urine incontinence
clonic مع 35
منظري 12 م
relax

تايم منك لما فاقه
due to Brain hypoxia
due to Resp Muscle strain

الفاروق
تباع 4
انه حنانه
unilat.
Bilat

اهم دواعي

associated
loss of body tone

staring
gazing

اشهر الاسباب واحدة في جهة الارباعي
ما ساه غير حاجه اجابتي حاجات قبله attack عند الشللة
White Knight Love

myoclonic 3-times ← aura of grand mal
petit mal
لوحده

III. Myoclonic Seizures: "attacks of involuntary clonic movements"

Onset: sudden
Offset: sudden

- It is characterized by: sudden, jerky, shock-like INVOLUNTARY muscle contraction.
 - The jerks are bilateral contractions, mainly of the shoulders and arms.
 - However, some patients report jerking in the lower limbs, trunk, or head.
- It may be of 2 types:
 - Simple: - Occurs singly (no loss of consciousness).
 - As a part of: - Grand mal epilepsy (aura). - Petit mal epilepsy.

Drug of choice: Valproate (Depakine) or Clonazepam (Rivotril)

IV. Atonic seizures: V.V.V. rare

- Transient attacks of brief loss of postural tone, often resulting in falls and injuries.

2. PARTIAL SEIZURES

"Excessive electrical discharges from cortical neurons in a certain area in ONE hemisphere"

A. Simple seizures:

"No disturbance in consciousness"

- The CP depends on the site of the hyperexcitable neurones in the cerebral cortex, whether in: "Motor area or Sensory areas".

1. Motor fits:

- Focal fits:
- Motor jacksonian fits:

2. General Sensory fits:

- Focal fits:
- Sensory jacksonian fits:

3. Special Sensory fits:

- Visual hallucinations:
- Auditory hallucinations:
- Olfactory hallucinations:

movement of part of a limb or the whole limb.
movement of one side of the body (see before).

parasthesia of part of a limb or the whole limb.
parasthesia of one side of the body (see before).

irritation of the visual sensory area.
irritation of the auditory sensory area.
irritation of the uncus.

B. Complex seizures:

"disturbance in consciousness"

SITE: The hyperexcitable neurons are in the Temporal lobe "Temporal lobe epilepsy".

DURATION: The seizure lasts few seconds to few minutes.

The seizure starts with Aura, followed by Absence, Automatism, Amnesia:

1. **Aura:** derealization, déjà-vu phenomenon, Sensation of fear. (already seen)
2. **Absence:** Absent patient with staring eyes (with no response to conversation).
3. **Automatism:** Involuntary Purposeless acts: motor (eg, lip smacking, chewing,) or verbal.
4. **Amnesia:** No recalling of the seizure. recent event

no motor auras
no general sense

Smelling recent memory behaviour

White Knight Love

3. PARTIAL SEIZURES → GENERALISED SEIZURES

"Partial seizures may spread to involve the whole brain → secondarily generalised seizures".

Psychogenic Epilepsy

Hysterical

- The cause is psychological & there is no organic lesion.
- ↑ • It usually occurs in the presence of people.
- ↓ • It never occurs during sleep.
- The patient never hurts himself: e.g. there is no tongue biting.
- There is no incontinence of urine.
- The EEG is normal.

حالة غير عضوية
organic

4

PRECIPITATING FACTORS

- Missed AED → stimulates GABA [فانغ بروج-شغل]
- Menses.
- Alkalosis. $\uparrow \text{pH} \rightarrow \downarrow$ threshold of brain cells stimulation
- Alcohol use & Drug abuse (e.g. cocaine). (excitatory)
- Stimulation by photons & Hyperventilation.
- Sleep deprivation & Stress & sudden withdrawal of antiepileptic drugs.

Causes disturbance of balance between Excitatory & inhibitory in favor of excitatory

لأنه يسهل إثارة

أثناء العلاج
عاز الحماض
يكون على حافة
acidotic
عشاق يرفع
threshold

5

INVESTIGATIONS

1. EEG:

- It is the most specific test for epilepsy because it records the electrical activity of the brain.
- It shows specific pattern: "Epilepsy waves".

2. LOCAL INVESTIGATIONS:

"CT & MRI of the brain"

- To identify or exclude a LOCAL CAUSE of seizures in the brain.

eg: Hge or tumor

3. GENERAL INVESTIGATIONS: "Laboratory investigations"

- To search for a GENERAL CAUSE of seizures, e.g. blood glucose.

↓ Ca

6

TREATMENT

A. General Measures:

1. Moderation of the patient's physical activity.
2. Avoid the precipitating factors (Alcohol, hyperventilation, photic stimulation.....).
3. A ketogenic diet is encouraged because it will induce acidosis:
- Acidosis is beneficial as it raises the threshold of stimulation of the brain cells.

B. Specific Treatment:

1. Treatment of the cause in secondary epilepsy. eg Control
2. Anti-epileptic drugs:
 - a) Always start with one drug, then add another drug if there is no response.
 - b) Always stop the drugs ONLY if:
 - The patient stays free of symptoms for at least 2 years.
 - The patient has a normal EEG.
3. Side effects of Anti-epileptic drugs:
 - 1. Skin rash.
 - 2. Bone marrow depression. (aplastic anemia)
 - 3. Ataxia. toxic on cerebellum (years)
 - 4. Drowsiness

ANTI-EPILEPTIC DRUGS

Drug	Dose	Best indicated
1. Barbiturates: Luminal (Phenobarbitone)	100-600 mg / day	- Broad spectrum. - Not for petit mal.
2. Hydantoin: = phenytoin Epanutin	100-600 mg / day	- Grand mal. - Motor Jacksonian fits.
3. Carbamazepine: Tegretol	200-600 mg / day	- Grand mal. - Motor Jacksonian fits. - Complex seizures. - Not for petit mal.
4. Clonazepam: Rivotril	2 - 6 mg / day	- Myoclonic. - Grand mal.
5. Valproate: Depakine	500-1500 mg / day	- Broad spectrum.
6. Succinamide: Zarontin	500-1000 mg / day	- Petit mal.

NEW ANTI-EPILEPTIC DRUGS

- These drugs are new drugs that may be used in resistant seizures.

1. Lamotrigine: 200 - 400 mg / day.
2. Felbamate: 400 - 800 mg / day.
3. Gabapentin: 600 - 1200 mg / day.

gradually stop drugs
- anti-epileptic
- anticoagulant
- cortisol



STATUS EPILEPTICUS

DEFINITION

- A medical emergency where there are:
 1. Repeated attacks of **generalized convulsions**, with **lack of recovery of consciousness**,
 2. **Persistent attack of seizure lasting for at least 30 minutes.**
- If the convulsions are not stopped rapidly, **coma deepens** & death may occur due to: **heart failure** or **respiratory failure** or **brain damage** or **hyperpyrexia**.
- The most common causes are: **sudden withdrawal of anti-epileptic drugs** & **stroke**.

TREATMENT

A. General Measures:

1. Take care of: **"ABC"**
 - Place the patient on the ground, to guard against falling from bed.
 - Mouth gag & O₂ inhalation (endo-tracheal intubation may be needed).
 - Record the vital signs regularly.
2. Take a sample of:
 - Venous blood: for the level of: **anti-epileptic drugs, alcohol.**
 - Arterial blood: for the level of: **pH, pO₂, pCO₂, HCO₃.**

3. Give cerebral dehydrating measures: e.g. **Lasix, conc. Mannitol, Dexamethazone.**

B. Specific Treatment:

- **Epanutin with Valium (or Rivotril) are given immediately:**

1. **EPANUTIN** (Phenytoin): 15 mg / Kg slow infusion.
 2. **VALIUM** (Diazepam): 5 mg slowly IV, to be repeated after 5 minutes if seizures recur: maximum dose: 20 mg.
- OR:
- RIVOTRIL** (Clonazepam): 2 mg slowly IV, to be repeated after 5 minutes if seizures recur: maximum dose: 6 mg.

- **If seizures persist after 20 min. of Epanutin & Valium:**

3. **PHENOBARBITONE:** 200 mg infusion.

- **In resistant cases:**

4. **GENERAL ANAESTHESIA:** may be used.

- تستخدم في هذا الكتاب
- uses
1. stroke (↓ edema)
 2. Bell's Palsy (canal)
 3. GBS
 4. Myasthenia
 5. MS
 6. Meningitis

HEADACHE

نظري
Causes of Headache

DEFINITION

Mild to severe pain in the head including the **FRONT** of the face & the **BACK** of the head.

6. cont'd 5. B-opsy from A. 4. tender scalp (vasculitis) & Blindness 1. disease of large B.V. especially Superficial Temporal A.

ETIOLOGY & TYPES

1. Vascular headache:

due to **Vascular dilatation** or **Vasculitis**

a) Primary:

MIGRAINE,

Cluster headache, Temporal arteritis.

b) Secondary:

- Hypertension.
- Hypoxia.
- Hypoglycemia.
- Drugs causing VD:
- Toxins causing VD:

e.g. Nitrates.

Alcohol,

Infections

(bacterial or viral toxins).

2. Inflammation:

- Intracranial:
- Neuritis:

Meningitis, Encephalitis.

of the sensory nerves of the scalp: e.g. Trigeminal neuralgia.

3. Traction headache:

due to stretch of the meninges, e.g. ↑ICT

- Brain tumour, abscess, or hemorrhage.

- Post-lumbar puncture.

4. Muscle contraction headache:

When one gets a "STIFF NECK," the prolonged muscle spasm can compress the nerves and also induce ischemia with production of metabolites which stimulate pain receptors:

- Prolonged driving.

- Cervical spondylosis.

5. Referred headache:

due to diseases in different parts of the head

- Eyes:
- Ears:
- Nose:
- Mouth:

- e.g. glaucoma.
- e.g. ear infections
- e.g. sinusitis
- e.g. dental sepsis.

6. Tension headache (Psychogenic):

"the most common cause"

- It occurs in:
- It occurs as:

psychologically disturbed persons usually FEMALES.
pressure or BAND-LIKE, Bilateral fronto-occipital.

Oral 1000

CRITERIA OF A SERIOUS HEADACHE

1. SUDDEN, severe headache. *A. → ruptures suddenly*
2. Headache associated with projectile vomiting & blurring of vision: *worst headache - life (i.e. SAH)* **↑ ICT.**
3. Headache that begins after an INJURY to the head. *Extradural*
4. Headache followed by DCL or associated body weakness. *subdural*
5. Headache associated with FEVER and a stiff neck. *لحم العج*

disturbed consciousness level

*1 * Meningitis
2 * SAH*

Stroke (hemiplegia)

MIGRAINE

*hge oi
jav se
disturbing in Heat
Regulatory
Center*

DEFINITION

- It is a paroxysmal disorder characterized by intense throbbing headache, (usually unilateral) associated with autonomic manifestations, e.g. nausea & vomiting.

ETIOLOGY

- Idiopathic.
- GENETIC:

Heredo-familial (80 %).

INCIDENCE

- SEX:
- RESIDENCE:
- PERSONALITY:

more in young FEMALES (female / male = 3 / 1).
more in urban areas.
more in perfectionistic people.

MIGRAINE TRIGGERS

"4 M"

- **M**ental & physical exertion.
- **M**enses.
- **M**edications:
- **M**eals:

*Vasodilators (nitrates), CCPs.
Cheese, Chocolate, Citrus fruits.
Smoking, Alcohol.*

CRITERIA OF A SERIOUS HEADACHE

1. **SUDDEN**, severe headache. *A. → ruptures suddenly worst headache - life (ie. SAH)*
2. Headache associated with projectile vomiting & blurring of vision: **↑ ICT.**
3. Headache that begins after an **INJURY** to the head. *Extradural subdural*
4. Headache followed by **DCL** or associated body weakness. *لازم العرج يفترج*
5. Headache associated with **FEVER** and a stiff neck. *Stroke (hemiplegia)*

MIGRAINE

DEFINITION

- It is a paroxysmal disorder characterized by intense throbbing headache, (usually unilateral) associated with autonomic manifestations, e.g. nausea & vomiting.

ETIOLOGY

- Idiopathic. *Unknown etio (but well known pathophysiology)*
- GENETIC: *Heredito-familial (80%). runs in families*

INCIDENCE

- SEX:
- RESIDENCE:
- PERSONALITY:

more in young FEMALES (female / male = 3 / 1).
more in urban areas. *stress*
more in perfectionistic people. *Type A stress*

MIGRAINE TRIGGERS

- **M**ental & physical exertion.
- **M**enses.
- **M**edications: *eg Vasodilators (nitrates), CCPs.*
- **M**eals: *eg Cheese, Chocolate, Citrus fruits. الموالج*

Smoking, Alcohol. *المكبات*

• Alcohol *أخضر*

- PN
- Myopathy
- Wernick's encephalopathy
- Headache
- Migraine *White Knight Love*

* مثل لازم أبداً قبل migrain تأتي aure ده بالعكس غالباً لا تأتي قبله أنه لما اتوصفت كلاسيفي في الأصل *
 (Classic not usual (not common))
 diagnosed only clinically — (MCO) 138
 بالترجمة، إقليمية، إيلي مالاش. investig.

aure: also in epilepsy

CLINICAL PICTURE

2 stages

1. First stage:

"Aura"

due to VC

- Visual: (17) Flashes of light or blind spots.
- Sensory: Parasthesias. tingle, numbness, burning
- Motor: Weakness.

sudden transient attack of VC

sudden transient ischemia (at any area of brain)

sudden transient disturbance of fn. [Aura]

2. Second stage:

"Migraine"

due to VD

It occurs in periodic & recurrent attacks:

- SITE & REFERENCE: Starts in the temple or around the eye & then: Spreads to involve the whole side of the head.
- CHARACTER: Pulsating or Throbbing.
- DURATION: It lasts for few hours to few days (3 hours to 3 days).
- AGGRAVATED BY: Bright light, Physical & Emotional stress, noise
- RELIEVED BY: Sleep.
- ASSOCIATIONS: Nausea, vomiting, Photophobia, Phonophobia.

PATHOGENESIS

1. NEUROVASCULAR THEORY

A) VASOACTIVE NEUROPEPTIDES

- Migraine triggers → abnormal overexcitation of Trigeminal nerve axons (Scalp) → release of VASOACTIVE NEUROPEPTIDES including Substance P from Trigeminal nerve endings (near the meningeal blood vessels).

B) VASODILATATION + STERILE INFLAMMATION

- These peptides will cause: VD + STERILE INFLAMMATION at the Trigeminal nerve endings that spreads to the nearby meninges. (giving serotonin agonist ⇒ potentiate action of serotonin in Brain ⇒ Block substance P)

C) PAIN results due to both VD & INFLAMMATION.

- ↑ Conduction of Pain by Substance P

Serotonin through acting on its receptors appears to block these peptides

2. SEROTONIN THEORY:

Initially: An initial increase in plasma serotonin levels will cause constriction of cerebral blood vessels and a reduction in cerebral blood flow → AURA.

Later: A subsequent drop in plasma serotonin levels will then lead to marked dilation of the arteries → HEADACHE of MIGRAINE.

لا تتركه الشفوي
تكتبه النظري آخر واحد

3. VASCULAR THEORY "OLD"

نرماله أي كلام
serotonergic excitatory neurotransmitter

Initially: An unexplained VC (hypoperfusion) begins in the visual area and spreads in the cerebral cortex for a short time → AURA.

Later: Reactive VD follows → HEADACHE of MIGRAINE.

4. DOPAMINE THEORY

excitatory neurotransmitter

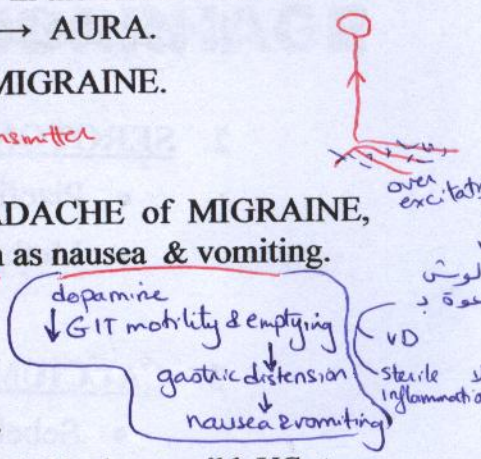
Recently: abnormal Dopaminergic stimulation → HEADACHE of MIGRAINE, and also causes associated manifestations such as nausea & vomiting.

: dopamine antagonist

TYPES

1. CLASSIC migraine (15 %): always preceded by aura.
2. COMMON migraine (85 %): not preceded by aura; probably due to mild VC stage.
3. RARE VARIANTS:

- Hemiplegic migraine: associated with transient hemiplegia.
- Facial migraine: associated with transient facial paralysis.
- Ophthalmoplegic migraine: associated with transient ophthalmoplegia.



TREATMENT

أهم علاج في الحصة

((ما يحلل الحياء ب attack يبقى VD))

oral pr

A. During the attack:

1. SEROTONIN AGONISTS:

They act on the serotonin receptors in the cerebral blood vessels (which were already dilated) leading to their VC & relief of headache.

الاحسن

a) Selective agonists:

SUMATRIPTAN → only on Brain B₁ receptors

- Orally: Single tab (50 or 100 mg), may be repeated after 2 hours, Maximum daily dose: 200 mg.
- Parentrally: 6 mg, single SC injection, may be repeated after 2 hours, Maximum daily dose: 12 mg (2injections).
- Nasal spray.

أهم
الادوية
بالفحاح
- DM
- DI
- migraine
- B.A.
- osteoporosis

b) Non-Selective agonists:

ERGOTAMINE

- Orally: Ergotamine, 1 mg, 2 tablets once. Ergotamine 1 mg + Caffeine 100 mg (added caffeine will enhance absorption of ergotamine & potentiate its VC effect).

2. DOPAMINE ANTAGONISTS:

METOCLOPRAMIDE (orally, IV)

- Adjunctive therapy in an acute attack of migraine. → ↓ excitation of nerve endings as ↓ dopamine
- Relieves associated nausea & vomiting (antiemetic effect).

3. ANALGESICS:

NSAIDs.

Prokinetic
يعمل
intestine

DD of any neurological disease

oral / نظري / clinical & case

DISSEMINATED SCLEROSIS (DS) MULTIPLE SCLEROSIS (MS)

DEFINITION

- It is an **INFLAMMATORY** disease of the CNS (Brain & Spinal cord).
- It affects predominantly the **WHITE MATTER** in the form of patchy **DEMYELINATION**.

ETIOLOGY

1. **AUTO-IMMUNE:** CSF shows increase in immunoglobulins especially IgG.

- ANTIBODIES** occur against proteins in the myelin sheath surrounding the nerves.
- This causes inflammation and injury to the sheath and ultimately to the nerves.
- The result may be multiple areas of scarring (**SCLEROSIS**).
- The damage slows down the nerve signals leading to impairment of the function.

2. **Less accepted old theories:** Viral infection or Ischemia of the white matter.

RISK FACTORS

- INHERITANCE:** The disease runs in families.
- INFECTIONS:** Viral or Bacterial infections may trigger the disease.
- Pregnancy & Labour:**
- STRESS:** Physical & Emotional.
- SURGERY & TRAUMA.**

CLINICAL PICTURE

TYPE OF THE PATIENT

Age: 20-40 years.

Sex:

more common in FEMALES.

BEHAVIOUR OF THE DISEASE

Onset: usually acute.

Course:

REMISSIONS & EXACERBATIONS.

not in 100%
in 75%

FEATURES:

any of the following manifestations:

1. Mentality changes:

- Euphoria or Depression or Emotional lability
- Cognitive dysfunction (memory loss).

2. Speech disturbance (Dysarthria):

- Staccato speech
- Slurred speech.

3. Cranial nerve affection:

- Optic: 2
- Oculomotor: 3
- Facial: 7
- Cochleo-vestibular: 8

Optic neuritis, ending in BLINDNESS.

Ophthalmoplegia with diplopia & squint.

UMNL (commonly), LMNL (rarely).

Vertigo.

4. Motor system affection

- Monoparesis, Paraparesis, hemiparesis, quadriparesis,
- Pseudo-bulbar palsy.

5. Sensory system affection

- Initially: parasthesias, Late: sensory loss (S or D) & sensory ataxia.
- L'hermite sign: Electric-like sensation felt in the back & limbs on bending the neck. It is due to posterior column affection in the cervical region.

6. CEREBELLAR AFFECTION:

- Features of cerebellar ataxia.

7. Autonomic affection:

- Sphincteric troubles: precipitancy or retention.
- Impotence.

Oral in clinical 1000

CLINICAL TYPES

1. Benign:

eg if motor: grade 4 or 3 only weakness 10 %

Symptoms are mild to moderate, do not worsen and do not lead to permanent disability.

2. Relapsing remitting:

75 %

One or two flare-ups occur every few years, followed by periods of remission. Flare-ups typically appear acutely, last a few weeks or months, then gradually disappear.

3. Progressive:

15 %

• Primary progressive:

After symptoms first appear, continuous deterioration occurs without periods of remission.

• Secondary progressive:

After years of relapsing remitting MS, continuous deterioration occurs in half the patients.

EARLY MANIFESTATIONS

1. EYE:

Optic neuritis.

2. AUTONOMIC:

Sphincteric troubles & Impotence.

as relapsing remitting but:

no remission

WhiteKnightLove

INVESTIGATIONS

1. Fundus examination:

- Pallor of the optic disc, especially on the temporal side.

2. CSF examination:

- Cells: Marked increase up to 50 cells / mm² mainly lymphocytes.
- Proteins: Moderate increase.

The most sensitive & specific findings in CSF

- Increase in **IMMUNOGLOBULIN** concentration, especially IgG.
- Presence of **OLIGOCLONAL BANDS** on protein electrophoresis.

3. Cortical Evoked Responses:

"CER"

Normally: Stimulation of any sensory receptor (visual, auditory, or somatosensory) evokes an electrical signal in the corresponding region of the cerebral cortex "CER".

In MS: Stimulation of any sensory receptor (visual, auditory, or somatosensory) evokes a slow or abnormal CER due to loss of myelin (↓ nerve impulse conduction).

Therefore: Recording of CER may help in detection of demyelinated lesions in MS.
e.g. Abnormal Visual Evoked Potential (VEP) = lesion in the visual pathway.

4. IMAGING:

I. MRI:

A. Confirms the diagnosis:

Patchy multiple areas in the white matter (areas of demyelination).

B. Differentiates new lesions from old lesions: **MRI with Gadolinium**

In new lesions of MS, recent inflammation leads to increased vascular permeability; this is detected by leakage of IV contrast agent Gadolinium into the brain on MRI.

II. CT scan:

Patchy multiple areas in the white matter (areas of demyelination).

↓ frequency (↑ remission)
 ↓ severity
 ↓ disability (↑ time of delay) morbidity

TREATMENT

No curative ttt

1. Immunomodulatory drugs:

"ABC"

Restore fn. of
 Immune system
 to Normal
 T cells

- A** Avonex: interferon β -1a.
B Betaseron: interferon β -1b.
C Copaxone.

α -interferon
 in H of CML &
 Hepatitis
 B & C

- They reduce: the frequency & severity of the attacks.
- They delay: the progression to disability.

2. Immunosuppressive drugs:

"MAC"

↓ immune system
 ↓ Abs formation

- a) **M**ethotrexate. b) **A**zathioprine. c) **C**yclophosphamide.

3. TTT of acute exacerbations:

I. Corticosteroids:

Give:

- a) Methyl prednisolone: 1 gm IV infusion / day for 1 week **OR:**
 b) ACTH: 80 IU given IM / day for 1 week, **then:**
 40 IU given IM / day for another week.

Continue with: Maintenance

- Prednisone: 1 mg / Kg / day orally for 1 week, **then:** taper gradually.

II. Plasmapheresis: may be of value in cases not responding to corticosteroids.

4. Symptomatic TTT, Physiotherapy & Vitamins.

Methyl prednisone
 ACTH
 Prednisone

1w 2w
 2w 3w
 1w

gradual withdrawal
 White Knight Love

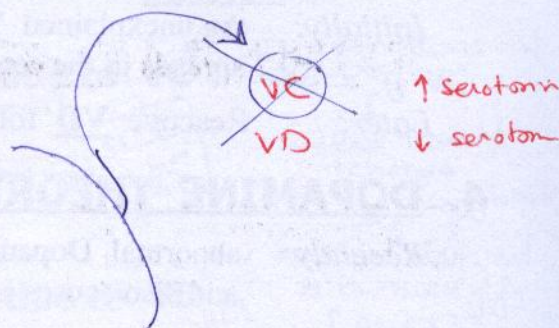
B. Inbetween the attacks:

"Prophylactic"

1. Avoid Migraine triggers.

2. SEROTONIN ANTAGONISTS:

- Pizotifen: 0.5 mg tds.
- Methysergide: 1 mg tds.



3. CALCIUM CHANNEL BLOCKERS: to avoid VC (aura)

- Sebelium: 10 mg / day before sleep.
- Verapamil: 80 mg tds.

4. BETA-BLOCKERS:

- Propranolol:

Action: It prevents the uptake of adrenaline by the receptors in the vessel wall.

Dose: 10 mg t.d.s.

Beta-blockers are the 1st of choice in patients whose migraine attacks are related to stress

as sympatholytic → ↓ stress → ↓ migraine attacks

5. OTHER MEASURES:

- Antihistaminics.
- Antidepressants.

MCQ Antiranginal drugs useful in migraine pt.

6. Antiepileptic eg: Gabapentin

some anti serotonin effect

also DD of facial Pain

↓ conduction

مجموعات زمنية

Cluster Headache

migraine is cluster cause

- **Character:** attacks of severe headache starting on one side in the orbital & frontal region & spreading gradually to the same side of the head & neck.
- **Duration:** several minutes to one hour.
- **Frequency:** the attacks occur in clusters, i.e. every 24 hours for weeks or months. the clusters are followed by long free periods (6 months or one year).

on weeks OFF
cluster 6M-1y cluster 6M-1y cluster

دورات متكررة

Q: Diagnosis

CIP complication

جاء بسنة قبل اللى فانت
نظري

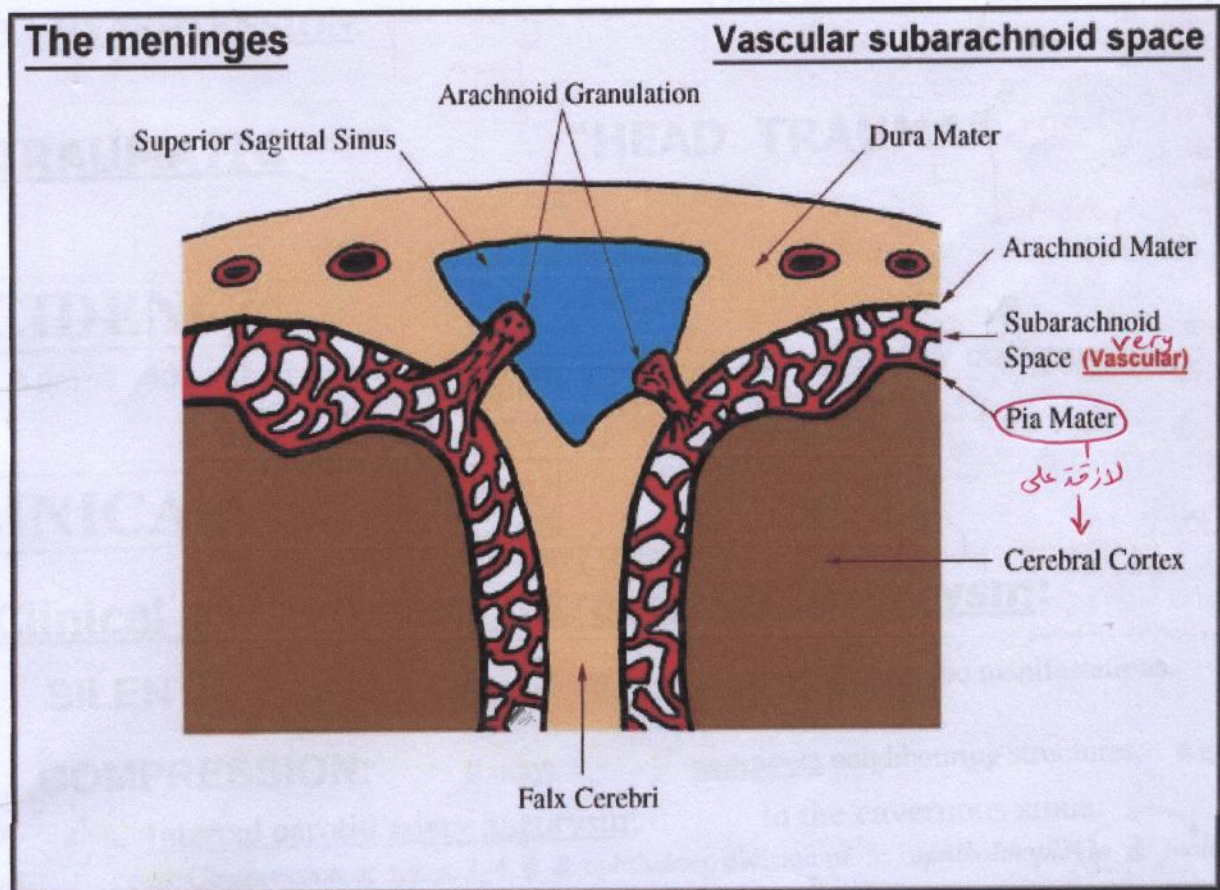
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SUBARACHNOID HAEMORRHAGE

DEFINITION

- The presence of blood in the subarachnoid space.

= [Flooding of " " Blood]



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ETIOLOGY

I. SPONTANEOUS

1. RUPTURE

- Intracranial aneurysm: **"most common cause"**
 - a. Congenital.
 - b. Atherosclerotic.
 - c. Mycotic (e.g. in SBE).
- Intracranial AV malformation **[congenital only]**

degenerated wall → weak

Rupture of Aneurysm or Athero

2. HEMORRHAGE

- Intracerebral Hemorrhage: that extends to the subarachnoid space.
- Hemorrhage in a brain tumour: that extends to the subarachnoid space.
- Hemorrhagic blood diseases: **purpura, hemophilia.**

3. HYPERTENSION.

mostly cause Intra cerebral SAH
may " ↓ platelets till 10,000 life threatening

II. TRAUMATIC

"HEAD TRAUMA"

mostly cause sub or extra dural
rarely → SAH

INCIDENCE

- Age: 40 - 60 in case of aneurysm. (atherosclerosis)
10 - 20 in case of AV malformation.

CLINICAL PICTURE

I. Clinical picture of an intracranial aneurysm:

1. SILENT:

It may:

remain **silent** with no manifestations.

2. COMPRESSION:

It may:

compress neighbouring structures, e.g.

a. Internal carotid artery aneurysm:

in the cavernous sinus:

- Compression of Cr. n. 3, 4, 6 & ophthalmic division of 5: **ophthalmoplegia & facial pain.**

b. Basilar artery aneurysm:

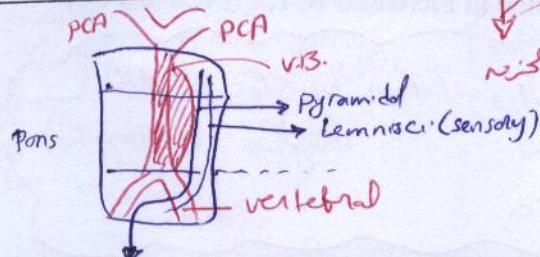
compression of midbrain or pons:

- Cranial nerve palsies & long tract manifestations (e.g. hemiparesis).
eg. sensory affection (lemnisci)

3. RUPTURE:

90% of cases first present when they **rupture** giving CP of SAH.

Rupture may be precipitated by: Strain, Stress, Severe cough or Sexual intercourse.



From its ligaments → SAH

WhiteKnightLove

II. Clinical picture of subarachnoid hemorrhage:

* 1. Features of INCREASED ICT:

a) Headache:

due to irritation of the meninges by blood

- Onset: SUDDEN.
- Character: SEVERE (**worst headache in the life**), ↑ by flexion of the neck.
- Site: STARTS at the back of the head & upper neck, THEN: generalized.
- Reference: Shoulders, back & limbs. due to all meninges by circulating CSF

b) Vomiting:

Projectile

c) Papilloedema.

* 2. Features of MENINGEAL IRRITATION:

a) Pain:

due to irritation of the spinal sensory roots

- In the back of the neck, shoulders & upper limbs.
- In the lower back & lower limbs.

b) Neck:

stiffness or rigidity.

c) Body:

opisthotonus (high arched back).

d) Positive signs of meningeal irritation.

POSITIVE SIGNS OF MENINGEAL IRRITATION

Kernig's sign: While the hip and knee joints are flexed at 90°, Extending the knee is limited & painful due to stiffness in Hamstrings.

Lasegue's sign: While the hip and knee joints are fully extended, Raising the leg by flexing the hip is limited & painful.

Brudzinsk's sign:

- Neck sign:** Passive flexion of the neck leads to flexion of both hips & knees.
- Leg sign:** Passive flexion of one hip leads to flexion of the other hip & knee.

3. Features of CRANIAL NERVE AFFECTION:

The most commonly affected nerves are the ocular nerves (3, 4, 6) & the optic nerve (2).

4. Features of EYE AFFECTION:

- Flame-like retinal hemorrhages:** due to blood in the subarachnoid sheath of optic n.
- Papilloedema:** due to increased ICT. (Brain edema)

These 3 tests are +ve in 3 only diseases

- meningeal irritation by Blood (SAH)
- " " pus (meningitis)
- Sciatica

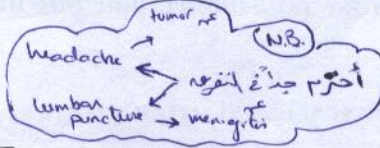
WhiteKnightLove

5. Features of CEREBRAL AFFECTION: *due to pressure by the blood*

- Confusion & Coma.
- Cortical sensory loss.
- Convulsions.
- Contralateral hemiplegia.
- Contralateral homonymous hemianopia.
- Aphasia.

Remember:

CORTICAL
HEMIPLEGIA



organised
hematoma
↓
pressure

Ischemia
due to
pressure
on
Blood
vessel

INVESTIGATIONS

1. CT scan or MRI:

the first investigation to be done

- If blood is detected, the diagnosis is established and lumbar puncture is not needed.

2. Lumbar puncture:

for CSF examination

- High pressure.
- High protein.
- NORMAL sugar and chloride.
- Grossly bloody.
- Culture is negative. (vs meningitis)

(meningitis ↓↓)

لأنه أنا عايز أوفرها
لا أعطي إلا إذا اضطرت لا
لأنه لا مائل كثير (invasive)
فلو imaging بيظهر نزيف لا تعجل
لأنه لو لم يظهر نزيف فاعجل
وده لانه C.T هنا 90% sensitive
10% sensitive 100% Intra cerebral

3. Cerebral Angiography: (localization) (done only if C.T +ve)

- Localises the site of the aneurysm or the AV malformation.

4. Plain X-ray skull:

- Calcified wall of aneurysm or AV malformation.
- Erosion or fracture of bone. Pressure ischemia by aneurysm
- Signs of increased ICT (in case of a brain tumour).

Sutures wide
silver (finger prints) if cause in
Brain tumor

COMPLICATIONS

1. Rebleeding:
2. Vasospasm: *in surrounding B.V.*
3. Hydrocephalus:
4. Epilepsy:
5. HYPONATREMIA:

associated with a worse prognosis.

causes cerebral ischemia or infarction.

due to occlusion of CSF flow by the blood.

due to cortical irritation and damage.

due to SIADH.

also in
1. Paraneoplastic Syndrome
2. Leukemia

irritative
lesion → hypothalamus

epileptogenic
focus

relative
↓ Na

↑ H₂O
retention

↑ ADH

if Fibrosed
& organised
in both
children & adult

oral (1) Reflex vaso-
spasm

لوحقت أجبلة
ACA
لأنه لو هذا
side effect 144
(تقولا مرة)

DIFFERENTIAL DIAGNOSIS

1. Other causes of coma. *eg: Stroke*
2. Other causes of cerebral hemorrhage.
3. Meningitis: *Intra-cranial* "Refer to DD of meningitis".
4. Differentiate: SAH from post-lumbar puncture traumatic blood.

	Subarachnoid hemorrhage	Post-lumbar puncture
1. Blood density	Constant in all samples.	Diminishes in successive samples.
2. Blood clotting	Blood will not clot.	Blood will clot.

TREATMENT

1. Medical:

INDICATIONS

1. Acute cases. *haemodynamically unstable*
2. Non-surgical cases, *e.g. hemorrhagic blood diseases. / HTN*
3. Difficult surgical cases, *e.g.*
 - Multiple aneurysms.
 - Huge or inaccessible aneurysms.

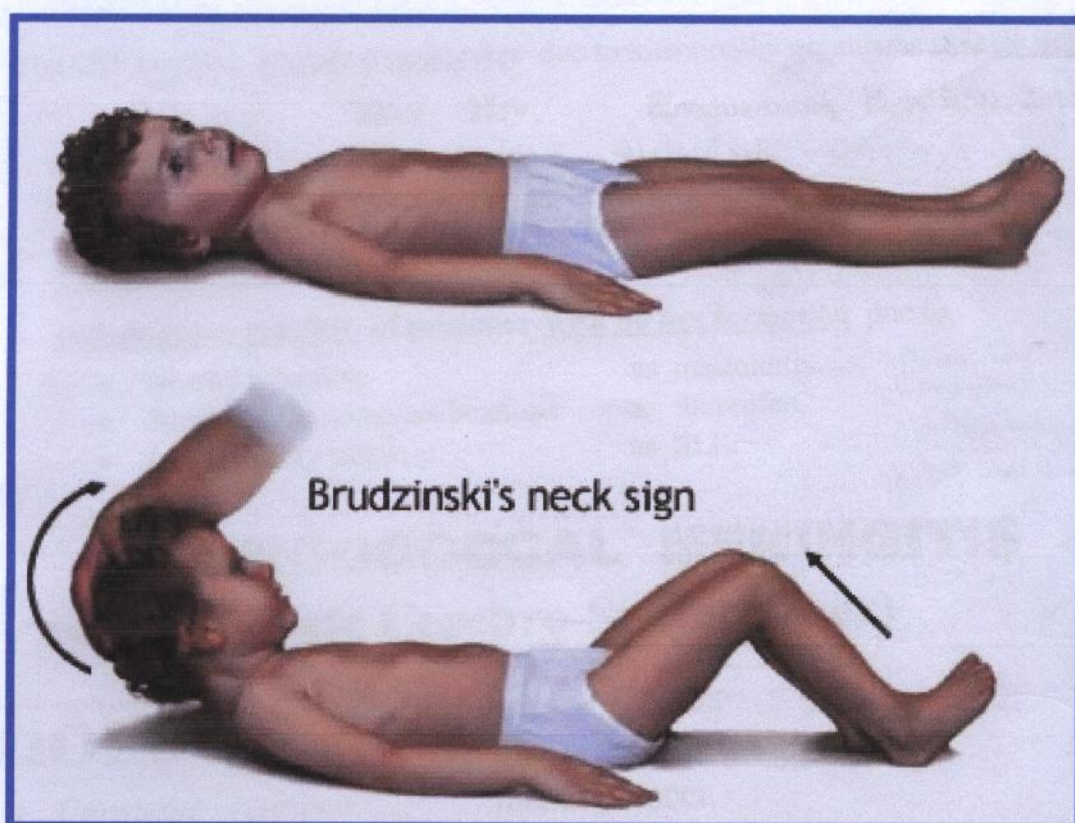
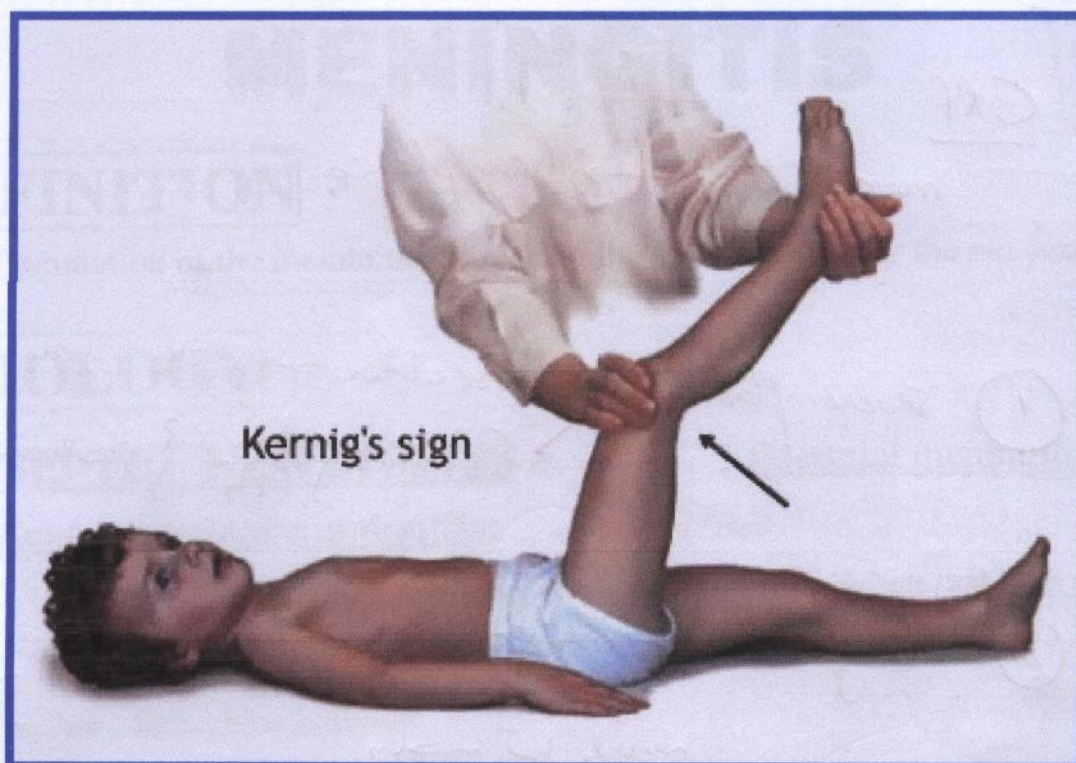
MEASURES *Conservative*

1. Avoid the precipitating factors for rupture.
2. Analgesics to relieve headache.
3. Antifibrinolytic agents: *e.g. aminocaproic acid*
 - They prevent clot dissolution around the aneurysm and thus prevent rebleeding.
4. Dehydrating measures for the brain: *eg cough control prophylactic to avoid rupture if not occurred* *e.g. IV mannitol.*
5. Lumbar puncture: *to relieve severe headache; should be done carefully*
6. Treatment of the cause: *e.g. ttt of Hypertensive emergency.*

2. Surgical:

- Direct surgery to the aneurysm or the AV malformation.

on top of diagnosis by cerebral angiography



infection → pus
non infective → no pus

MENINGITIS

نظري
oral

DEFINITION

- Inflammation of the membranes covering the CNS, especially the pia-arachnoid.

not infection viruses

ETIOLOGY

1 SEPTIC MENINGITIS

"Bacterial meningitis"

Acute purulent meningitis:

Infection ⊕ Pus

- The CSF contains mainly polymorphs due to infection by organisms that form pus:

- Meningococci.
- Streptococcus pneumoniae.
- Hemophilus influenza.

2 ASEPTIC MENINGITIS

"Non-Bacterial meningitis"

a. Subacute lymphocytic meningitis:

Infection ⊕ no pus

- The CSF contains mainly lymphocytes due to infection by organisms that do not form pus:

- Viruses: HSV, HIV, Enteroviruses, Echovirus & Coxsackie.
- TB: TB meningitis. → *من التهابات السحايا الفيروسية*
- Fungi: Cryptococcosis, Mucormycosis.

also in Bell's Palsy

b. Meningeal reaction:

NO infection ⊕ no pus

also in atypical pneumonia (erythema multiforme)

- Inflammatory reaction of meninges with no pus formation due to:

- Nearby infection: as mastoiditis. → inflammatory mediators
- Sensitivity to some medications: as ibuprofen. irritate meninges
- Inflammatory diseases: as SLE. infection may not

also
- dry pleura
- dry pericardium
- DCM
- (E) DM
- ...

MENINGOCOCCAL MENINGITIS

(Acute Cerebro-Spinal Fever)

meninges around

ETIOLOGY

1. Causative organism: meningococci.
2. Source of infection: nasopharyngeal carriers.
3. Mode of infection: droplet infection.

is Rifampicin

التهاب السحايا الحاد
التي تسببها
sp. and Brain
نكاح

I.P. = 4-5 d

(death) may occur & not
Reach clip of meningitis
acc. to immunity
virulence of strains
of meningococci

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PATHOGENESIS

GRF
IP

1. NASOPHARYNX:

After infection, the organisms localize in the **nasopharynx** causing: catarrhal inflammation. *Rhinorrhea, fever, pharyngitis*

2. BLOOD:

The organisms then invade the **blood** → short meningococcemia (hours) which may be severe and cause: the fulminating type.

3. MENINGES:

From the blood they reach the **meninges** → purulent exudates which collect in the subarachnoid space causing: meningitis.

4. The organisms may be localized in:

- The adrenals → acute adrenal failure. *Addison*
- The skin → *Papular* skin rash.

CLINICAL PICTURE

1. GENERAL signs:

- Pyrexia: Acute high fever ($39 - 40^{\circ}C$).
- Pulse: Tachycardia, except in cases of $\uparrow$ ICT (bradycardia). *due to fever*
- Photophobia: Oversensitivity to bright light. *irritation to root of optic N.*
- Papular skin rash.

2. Features of INCREASED ICT:

a) Headache:

- Onset: **ACUTE.**
- Character: SEVERE,

due to irritation of the meninges by infection

$\uparrow$ by flexion of the neck.

b) Vomiting:

Projectile

c) Papilloedema.

3. Features of MENINGEAL IRRITATION:

a) Pain:

due to irritation of the spinal sensory roots

- In the back of the neck, shoulders & upper limbs.
- In the lower back & lower limbs.

b) Neck: stiffness or rigidity.

c) Body: opisthotonus (high arched back).

d) Positive signs of meningeal irritation: Kernig's sign..... etc....

4. Signs of NEUROLOGICAL DEFICITS:

- Confusion or coma. *= confusion [Toxic effect on Brain]*
- Convulsions.
- Transient cranial nerve paralysis: due to exudation around the nerves.
- Focal neurological signs: e.g. **HEMIPLEGIA:** rare.

usually quadriplegia
meninges of skull

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في الصفحة الفاضية

WhiteKnightLove

GRF

meningioma $\rightarrow$ hemiplegia

① severe inflammation → vasculitis → stroke in one Δ tract
 cytokines { IL, PAF } prothrombotic

(2) asymmetric organization

نواحیه قبل، السانیه

Addisonian crisis

hypotension
hypoglycemia
electrolyte disturb.

IV cortisol

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CLINICAL TYPES

رکت مع CLP

1. Acute meningococcal meningitis.

"classic form"

2. Fulminating type: The patient dies from marked toxemia before meningeal symptoms appear:

i. Cerebral type: fever, coma & death.

as - highest virulence
- lowest immunity

ii. Adrenal type: Waterhouse-Freidreichson's syndrome:

- Massive bilateral adrenal cortical hemorrhage leading to Addisonian crisis & death.

Acute Adrenal failure

3. Chronic meningococcal meningitis.

Low virulence organism

1. more gradual onset
more prolonged course
2. CLP mild but persistent
3. - low grade fever
- minimal signs of meningeal irritation

COMPLICATIONS

1. Neurological: Hydrocephalus due to occlusion of CSF flow by the organized exudates.
2. Cardiac: Pericarditis & Endocarditis. → death
3. Eyes: Conjunctivitis, keratitis, iridocyclitis, & may be BLINDNESS.
4. Ears: DEAFNESS due to affection of the 8th cranial nerve.
5. Others: Arthritis, nephritis, orchitis.

INVESTIGATIONS

لحمية في السائل الشوكي
→ CSF (report for hospitalization)

1. CT or MRI of the brain:

to exclude subarachnoid hemorrhage.

2. CSF examination:

meningococci → Pus

may as it need surgical intervention on the spot

- a. Pressure: high. (pus)
- b. Aspect: turbid, purulent.
- c. Cells: markedly increased especially POLYMORPHS.
- d. Proteins: markedly increased.
- e. Glucose: markedly decreased.
- f. Chlorides: moderately decreased.
- g. Organisms: detected by Gram-stain or CSF culture.

if TB, viral lymphocytes

if TB
↓↓↓ Cl
↓ G

3. CBC:

- Leukocytosis: with shift to the left.

staff: immature

B.M. سبيل
خلية
غير ناضجة

4. Culture of the blood:

- The organism may be detected in many cases of blood culture.

Shift to Rt is Megaloblastic

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C/Typhoid

- 1- Step ladder fever
- 2- very severe headache
- 3- Constipation (enterica)

اشترجاج
نقطة
في
acute infection

ادرجت

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DIFFERENTIAL DIAGNOSIS

اشترجاج في
meningitis
DD HT

1. Subarachnoid hemorrhage:

- ONSET: Sudden headache (as Blood)
- CSF: CSF is bloody, CSF culture is negative, CSF sugar & chloride are normal.
- CT scan: reveals the hemorrhage. (sensitive 90%)

2. Encephalitis:

- Presence of parenchymal involvement, e.g. cerebrum, brain stem, cerebellum.

3. Acute infections with cerebral symptoms:

e.g. Typhoid affect GIT especially intestine

- 1 Absence of signs of meningeal irritation.
- 2 Characteristic CP of the acute infection. (Typhoid)

4. Meningism:

(BBB)

MENINGEAL IRRITATION

- Meningeal irritation may occur in acute infections due to increased water content of the CSF in an attempt to dilute the toxins.
- CSF: clear, no organisms, high pressure, DILUTED: low glucose & chlorides & proteins.

5. Other causes of meningitis:

- a) Other causes of acute septic meningitis: e.g. Strept. pneumoniae, H. influenza

b) Viral meningitis:

- Course: benign.
- CSF: clear, NORMAL chloride & glucose, HIGH protein & lymphocytes.

c) TB meningitis:

- ONSET: Gradual.
- TB: Features of TB toxemia, Features of a primary TB focus.
- MENINGITIS: Minimal signs of meningeal irritation.
- CSF: Typical CSF findings: see the table.

Typical CSF of TB meningitis

- Pressure: high.
- Aspect: cloudy, smoky due to Microscopic RBCs
- Cells: increased especially LYMPHOCYTES.
- Proteins: markedly increased.
- Glucose: moderately decreased.
- Chlorides: markedly decreased.
- Organisms: detected by ZN stain or Lowenstein culture.

اشترجاج في

meningitis

- fever
- ↓ consciousness
- neuro deficit
- meningeal irritate

- but
- neuro
- meningeal irritate

Blood BBB CSF
infects
toxins → ↑H₂O
↑ pressure

- 1- no. C اشترج
- 2- DD. هذه
- 3- HT: discuss anti TB

مكرر كثر
management
of TB meningitis

SAH	CSF
Bloody	turbid
—	polymorph
↑	↑
2	↓↓↓
2	↓
—	Gm

في
ما
ل
غامغ
في
nephritic
syndrome

150

R.F. - بجيرة كالم

- Anti TB
- leprosy (dapsone)
- antibiotic prophylactic (meningitis)
- curative (Brucella)

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TREATMENT

I. Prophylactic:

1. Isolation of the patient.
2. Chemoprophylaxis:
3. Immunoprophylaxis:

Rifampicin 600 mg bid for 2 days for contacts.

Meningococcal Live attenuated vaccine.

لا تكون already اتخرفت لحيان

Red urine
لونه احمر

في اعادة لينة يومية

Passive immunization

II. Curative:

1. Antibacterial treatment:

- Once meningitis is suspected we should start ttt immediately even before culture:

- Cefotaxime (Claforan): 4 gm / day IV, (3rd generation cephalosporin) (no oral form of it) OR
- Crystalline penicillin: 4 million units / 6 hours IV, OR
- Chloramphenicol: 100 mg / Kg / day in penicillin-sensitive patients.

obsolete
لا في مصر
من علاج
التيقود

ال
quinolone
اقوى زهم
منه

2. Corticosteroids:

- In severe cases as:
- In case of:

Waterhouse-Freidreichson's syndrome.

TB meningitis undercover of Anti-TB drugs.

side effect → Aplastic anemia (irreversible B.m. depression)
dose & non dose dependent

as replacement
given IV

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ENCEPHALITIS

DEFINITION

- Inflammation of the brain substance.

ETIOLOGY

I. Primary encephalitis:

primarily attack the CNS

1. Neurotropic viruses: attack the nerve cells

- Poliomyelitis. AHCs
- Rabies.

LMNL ^{لحمي، جف}
fasciculation ^{جف}

2. Pantropic viruses: attack the neurones

- Arthropode borne viruses.

yellow fever ^{Brain}
liver as well

II. Secondary encephalitis:

do not primarily attack the CNS

1. Viral:

Herpes simplex, ^{التهاب}

Herpes zoster,

Mumps.

2. Bacterial:

Typhoid, ^{State}

Bacillary dysentery.

3. Parasites:

Malaria. ^{مرض الملاريا}

shigella

4. Others:

(MYCOPLASMA, chlamydia, rickettsiae.)

Atypical

- 1 - GGA or fluffy cotton
- 2 - erythema multiforme
- 3 - ↓ consciousness

Pneumonia

CLINICAL PICTURE

1. Manifestations of parenchymal involvement:

patchy focal OR diffuse

- Cerebrum: coma, aphasia, convulsions, hemiparesis.
- Brain stem: cranial nerve palsies, long tract manifestations.
- Cerebellum: ataxia, dysarthria.

cortical is

Brain stem nuclei long tracts

Lat. lemniscus: sensory
Focal → hemi
diffuse → Quadri

2. Manifestations of slight meningeal irritation:

may be present.

3. Manifestations of viral infection:

- General: fever, headache, myalgia, etc....

- Specific: depending on the causative virus, e.g.

- - LMNL paralysis with fasciculations: Poliomyelitis.
- - Parotid swelling: Mumps.

LMNL = fasciculation

HSV

- { - Mouth ulcers + vesicular skin rash
- erythema multiforme
- Hepatitis

INVESTIGATIONS

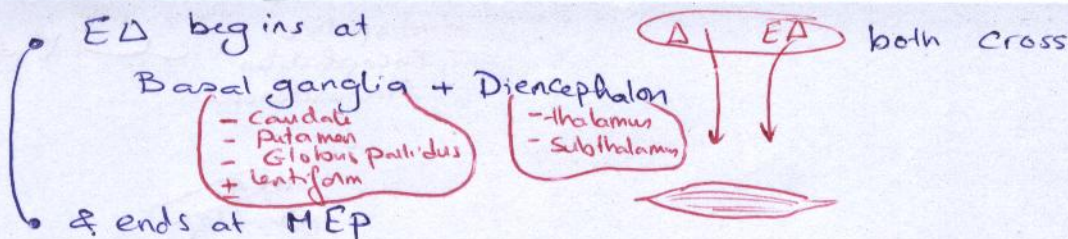
1. **MRI:** of the brain.
2. **EEG:** is helpful.

Not C.T.
as C.T. can't
diagnose
inflammation

TREATMENT

- Antiviral agent: Acyclovir. (for HSV)

Prognosis v. Bad.



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THE EXTRAPYRAMIDAL SYSTEM

DEFINITION

- It includes all fibres that can influence the MEP and do not pass in the pyramidal tract.

FUNCTION

1. **REGULATION** of the voluntary motor activity.
2. Regulation of the emotional & associated movements. → swinging hands on walking
3. Inhibition of the muscle tone. (not reflex) → Δ ↓ tone reflexes

DYSFUNCTION

1) **STATIC TREMORS** (disturbance in **REGULATION** of the voluntary motor activity):

- Regular: **Parkinsonism.** Both are involuntary movement
- Irregular: **Chorea, athetosis, dystonia.**

2) **BRADYKINESIA** (disturbance in **REGULATION** of the emotional & associated mov):

- Mask face (Immobile face with infrequent blinking.....staring look).
- Monotonous speech.
- Loss of swinging of the arms during walking.

3) **Rigidity (Hypertonia)** (disturbance in inhibition of the muscle tone).

PARKINSONISM (Shaking Palsy)

DEFINITION

- It is a condition in which there are **Static tremors (regular)** associated with: **Bradykinesia** & **Rigidity (hypertonia)** of the muscles of the body.

ETIOLOGY

I. **Idiopathic:**

- Age: Above 50 years.
- Sex: Both sexes are equally affected.

Deficiency of Dopamine in the Basal Ganglia

(paralysis agitans = unknown cause)

dopamine agonist

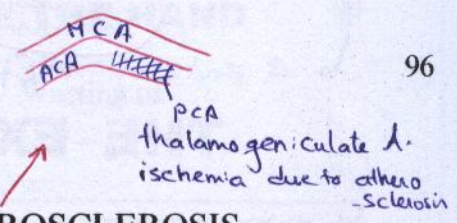
li 95

Migraine

dopamine

White Knight Love

- idiopathic
- Encephalitis
- Atherosclerosis



II. Secondary:

- | | |
|------------------|--|
| 1. INFLAMMATORY: | ENCEPHALITIS. |
| 2. VASCULAR: | CEREBRAL ATHEROSCLEROSIS. |
| 3. Toxic: | Major tranquilizers (<i>Phenothiazines</i>). |
| 4. Traumatic: | Repeated trauma to the head as in boxers. |
| 5. Tumours: | of the basal ganglia. |

الوردي
طويل

CLINICAL PICTURE

1. Static Tremors:

- Regular and occur at the rate of: 4-8 second.
- They give the hand the pill-rolling appearance.

as voluntary movement by Pyramidal
علاج

These tremors increase with: emotional stress & fatigue.
These tremors decrease with: sleep and during active voluntary movements.

2. Bradykinesia: Loss of emotional and associative movements

- Mask face (Immobile face with infrequent blinking.....staring look).
- Monotonous speech.
- Loss of swinging of the arms during walking.

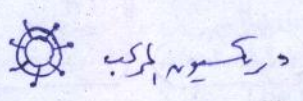
3. Rigidity: Hypertonia

A) Distribution:

- Proximal more than the distal muscles.
- Flexors of the neck, trunk & limbs resulting in the: *gorilla-like attitude*.

B) Character:

- Lead pipe: present throughout the act to the same degree, or:
- Cog wheel: interrupted by the tremors.



C) Gait:

- Short steppage: slow shuffling (Difficulty in starting the act of walking).

of V.B. Artery
Decerebrate

	Rigidity	Spasticity
Site of lesion	- Extra-pyramidal.	- Pyramidal.
Distribution	- Proximal more than distal. - Flexors of ULs - Flexors of LLs	- Distal more than proximal. - Flexors of ULs, } <i>antigravity</i> - Extensors of LLs.
Character	- Lead pipe or cog wheel.	- Clasp knife
Deep reflexes	- Normoreflexia or Hyporeflexia.	- Hyperreflexia.

Extensor UL
Extensor L.L.

⊕ MS power normal weak
⊕ Gait shuffling Circumducting
difficult although rigidity > tremors
due to severe stiffness & rigidity
may reach clonus
not smooth due to interruption by tremors
tremors > rigidity

The most common causes of Parkinsonism are:

- 1) Paralysis agitans. 2) Post-encephalitic. 3) Atherosclerosis.

Features	Paralysis agitans	Post-encephalitic	Atherosclerosis
Age	Above 50 years	Any age	Above 50 years
Onset	Gradual	Usually <u>acute</u>	Gradual
Course	Progressive <i>الهزة تزداد سنياً</i>	Regressive or stationary	Remissions & exacerbations
Rigidity	Tremors more than rigidity <i>الهزة أكثر من التصلب</i>	Rigidity & tremors equally marked	Rigidity more than tremors <i>head pipe</i>
Possible associated features		Diabetes insipidus <i>itis → of hypothalamus</i>	Hypertension Coronary heart disease Diabetes mellitus <i>insulin resistance</i>

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في بعض الحالات
الأثر
التهتك

due to
open of
collaterals

due to
atheroma
ischemia

insulin
resistance

TREATMENT

Q: Antiparkinsonial drugs

I. Medical

- The manifestations of Parkinsonism are due to an imbalance between the levels of:
↑ **acetylcholine** & ↓ **dopamine** in the basal ganglia.
- Reduction of dopamine and relative increase in acetylcholine levels are found in all cases of Parkinsonism, whatever the cause.
- Therefore medical treatment aims to restore the balance by:
 - Decreasing acetylcholine
 - or:
 - Increasing dopamine levels.

1) Anticholinergic drugs:

- a. Natural Belladonna alkaloids are rarely used nowadays. They include:
- Atropine sulphate.
 - Hyoscine.

b. Synthetic Belladonna-like alkaloids:

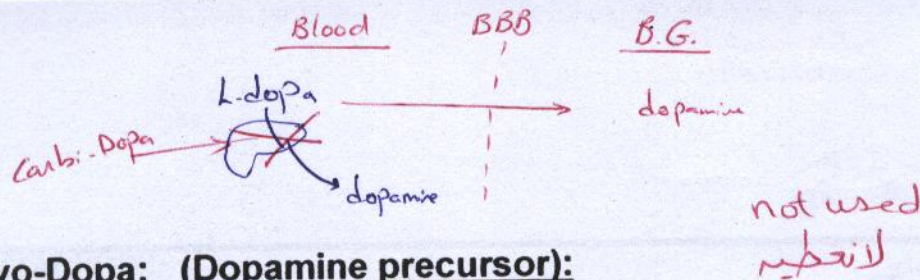
- * Cogentin 2 mg tab.
- * Artane 2 and 5 mg tab.
- * Parkinol 2 and 5 mg tab.

Dose: 1-2 tab. t.d.s., according to the severity of the case.

Side Effects: blurring of vision, dryness of mouth, retention of urine.

Toxic Effects: confusion and hallucinations.

Anticholinergics are best indicate in: TREMORS



2) Levo-Dopa: (Dopamine precursor):

- As dopamine does not cross the BBB, its precursor levo-dopa, which can cross it, is used instead.

Dose: $\frac{1}{2}$ g orally daily; increase it by $\frac{1}{2}$ g every 3 days. Max. dose: 4-6 g daily.

Side Effects:

- 1) Psychiatric: depression, confusion.
- 2) Cardiovascular: palpitations, arrhythmias.
- 3) Gastrointestinal: nausea, vomiting.
- 4) Neurological: chorea, hypotonia, epilepsy.

3) Levo-Dopa + Carbi-Dopa: (Sinemet)

- It was found that L-dopa is to some extent decarboxylated in the liver to dopamine before crossing the BBB resulting in:

1. The appearance of side effects due to the peripheral action of dopamine.
2. The necessity of using large doses of L-dopa to insure the passage of an effective dose across the BBB.

- These disadvantages are reduced by the addition of **Carbi-dopa** which inhibits the extracerebral decarboxylation of L-dopa to dopamine.
- Carbi-dopa itself does not cross the BBB.

Dose: One tab. of Sinemet contains 250 mg L-dopa + 25 mg Carbi-dopa.

Start with one tab. daily & increase the dose by $\frac{1}{2}$ tab. every 3 days till the case improves.

Sinemet is best indicate in: BRADYKINESIA & RIGIDITY

4) Dopamine agonists:

- They mimic the action of dopamine at receptor sites:

○ Trivastal:

20 mg t.d.s in conjunction with Sinemet.

○ Bromocriptine:

2.5 mg daily in conjunction with Sinemet.

Bromocriptine:

1. Parkinsonism

2. Acromegaly

3. Hepatic encephalopathy

5) Amantadine hydrochloride:

- It prevents the uptake of dopamine by the neurones:

100 mg tab. t.d.s.

6) Muscle relaxants:

to minimize rigidity.

II. Surgical

In severe cases

- 1) Pallidectomy. *Globus pallidus*
- 2) Thalamotomy.

CHOREA

DEFINITION

Involuntary STATIC movements of any part of the body:

- Irregular.
- Sudden & Jerky. *sudden onset*
- Pseudopurposive. *offset*

ETIOLOGY

I. Herido-familial: Huntington's chorea.

II. Secondary:

1. Autoimmune: Rheumatic chorea. *altered antigenicity*
2. Infective: Post-encephalitic chorea. *antigenic similarity*
3. Vascular: Hemiballismus. *Hge of subthalamus*
4. Toxic: Chorea gravidarum.
5. Neoplastic: Tumours of the Basal Ganglia.
6. Metabolic: Hepatolenticular degeneration (Wilson's Disease). *disturbance of ceruloplasmin*
7. Idiopathic: Senile chorea. *lentiform nucleus of B.G. liver*

↑ Cu

also H.A.

RHEUMATIC CHOREA (Sydenham's Chorea)

- It is one of the major criteria of rheumatic fever. *وجع ازالى يتحرك فجأة*
- It may be associated with other rheumatic manifestations, but never with rheumatic arthritis.
- Age: 5-15 years.
- Sex: females more than males.

$RF < \frac{\sigma}{\phi}$ but chorea $\phi > \sigma$

CLINICAL PICTURE

1. Choreic movements:

a) Affect the proximal muscles more than the distal muscles:

- When the patient is asked to keep his tongue protruded and unsupported by his teeth, he is unable to do so, and quickly retracts it. *عيب في السيطرة*
- Grimacing, jerking of the shoulders, shaking of the hands and feet. *Braking*

b) They increase with emotional stress and disappear during sleep.

RF انقلح دلوقتي في كتاب لانه كل كتاب في رتبة امكانه

2. Hypotonia: *as facilitatory Fibers lesion $\rightarrow$ $\uparrow$ tone*

- Choreic hand (scaphoid or boat-shaped hand):

When the patient stretches his arms there is flexion at the wrist and overextension at the metacarpophalangeal and interphalangeal joints with fanning of the fingers.

MCP PIP DIP

3. Emotional instability:

- Sudden laughter or sudden crying.

also MS

TREATMENT

1. Classic treatment of Rheumatic fever:

- Complete rest in bed.
- Corticosteroids as prednisone:
- Acetyl salicylic acid:

*1 mg / Kg / day.
6-8 gm daily.*

*on 5th week for 8 weeks,
as Carditis may be associated*

2. Specific treatment of Rheumatic chorea:

- Reserpine : 1 mg t.d.s.
- Haloperidol: 3 mg t.d.s.
- Tranquilizers.

انقل هناك دلوقة

*neuroleptic $\downarrow$ excitability of neurones
 $\downarrow$ neuronal discharge frequency*

*Reserpine
Anti HTN*

THE CEREBELLUM

The cerebellum can be divided into 3 main parts:

1. Archicerebellum:

- **Function:**
 - Maintenance of Equilibrium.
- **Lesion:**
 - Disturbance of Equilibrium.

2. Neocerebellum:

- **Function:**
 - Coordination of the voluntary motor activity.
- **Lesion:**
 - Incoordination of the voluntary motor activity.

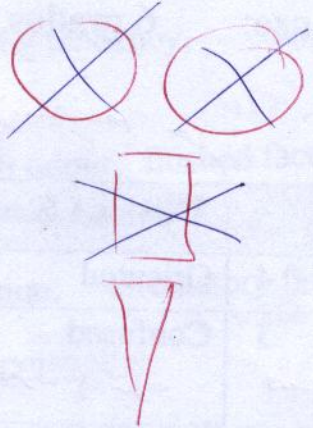
3. Paleocerebellum:

- **Function:**
 - Maintenance of the muscle tone.
- **Lesion:**
 - Hypotonia.

DeCerebrate
rigidity

قسط
brain stem
قشر
Cerebrum
sp. cord
Ext
Ext

↓
خيار
& deepest coma



Decorticate
rigidity
as Δ

hypertonia
[Flexors UL
Ext. L.L.

due to
removal inhibitory
impulse

for flexors : rubrospinal
for Extensors : vestibulospinal

2. Hypotonia: as facilitatory fibers lesion $\rightarrow$ $\uparrow$ tone

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MCP DIP
DIP

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also
MS

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1 mg / Kg / day.
6-8 gm daily.

on 5th week for 8 weeks
as candida may be associated

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- Haloperidol: 3 mg t.d.s.
- Tranquilizers.

انتقل هناك
دلوفا

neuroleptic

$\downarrow$ excitability of neurons
 $\downarrow$ neuronal discharge of impulses

Reserpine
Anti HTN

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The cerebellum can be divided into 3 main parts:

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- **Function:**
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- **Lesion:**
 - Disturbance of Equilibrium.

2. Neocerebellum:

- **Function:**
 - Coordination of the voluntary motor activity.
- **Lesion:**
 - Incoordination of the voluntary motor activity.

3. Paleocerebellum:

- **Function:**
 - Maintenance of the muscle tone. facilitatory to reflex
- **Lesion:**
 - Hypotonia.

Fibers
do not
cross

مسئ نظري
مسئ بشعوى
clinical n/w
short

مسئ لوصف
للم مع زاحفة
مسئ في حوض
(الديناميكية)
hypotonia only

ATAXIAS

DEFINITION

- Incoordination of voluntary motor activity with or without [±] disequilibrium in the absence of motor weakness.

TYPES

1. Cerebellar ataxia. 2. Sensory ataxia. 3. Vestibular ataxia. 4. Hysterical ataxia.

CEREBELLAR ATAXIA

ETIOLOGY

I. Heredo-familial:

1. Friedreich's ataxia. 2. Marie's ataxia.

II. Secondary:

- | | | | |
|---------------------|--|-------------------|-----------------------|
| 1. Infective: | - Encephalitis | - Meningitis | - Cerebellar abscess. |
| 2. Vascular: | - Superior, middle and inferior cerebellar artery occlusion. | | |
| 3. Toxic: | - Alcohol | - Antiepileptics. | |
| 4. Neoplastic. | - Paramalignant syndrome. , CPA tumor | | |
| ★ 5. Demyelinating: | - DS. | | |

most common cause



CLINICAL PICTURE

1. Incoordination of movements of different muscles in the form of:

1. The eye: Nystagmus.
2. The tongue: Dysarthria in the form of staccato speech.
3. The head: Nodding of the head.
4. The trunk: Titubation of the trunk (swinging from side to side).
5. The limbs: Intention kinetic tremors in the extremities.

رأس تهتز
جسم تهتز

2. Hypotonia and Hyporeflexia of the affected muscles.

3. Gait disturbance:

- In archicerebellar lesions: Drunken gait (wide-base gait).
- In neocerebellar lesions:
 - o In Unilateral lesions: Deviation of the body towards the affected side.
 - o In Bilateral lesions: Zigzag gait.

عشان اتزان ناقص

hypotonic
incoordination

4. Positive tests: used by the neurologist to detect cerebellar ataxia.

"HERIDO-FAMILIAL ATAXIAS"

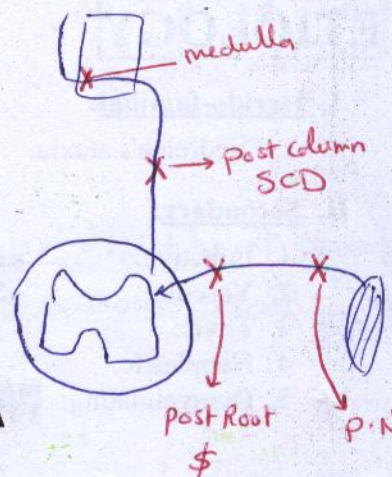
I. Friedreich's Ataxia

لا مركزا على
التي تتعلم

1. Age: it occurs in the 1st decade of life.
2. Onset & course: gradual onset and slowly progressive course.
3. Pathology: there is degeneration of:
 - Cerebellum especially the: archicerebellum.
 - Pyramidal tracts.
 - Posterior columns.
 - Peripheral nerves.
4. Associations: DCM, congenital heart disease, pes cavus.

II. Marie's Ataxia

1. Age: it occurs in the 2nd and 3rd decades of life.
2. Onset & course: gradual onset and slowly progressive course.
3. Pathology: there is degeneration of:
 - Cerebellum especially the: neo-cerebellum.
 - Pyramidal tracts.
4. Associations: Mental impairment.



SENSORY ATAXIA

DEFINITION

It is ataxia due to loss of the proprioceptive (deep) sensations, at any point in their pathway:

ETIOLOGY

- | | |
|----------------------|---------------------------------|
| 1. Peripheral nerve: | peripheral neuropathy. |
| 2. Posterior root: | tabes dorsalis. |
| 3. Posterior column: | subacute combined degeneration. |
| 4. Medial lemniscus: | brain stem lesions. |
| 5. Thalamus: | thalamic syndrome. |

CLINICAL PICTURE

1. Kinetic tremors as tested by finger-to-nose or finger-to-finger tests appear only on eye closure.
2. Rhomberg's test: when the patient stands with his feet close together & his eyes closed, his body sways & he may fall if not supported.
3. Stamping gait: heavy strike of the ground on walking due to lost deep sensation.
4. Deep sensory loss.
5. Hypotonia & hyporeflexia.

مشحنا بسعس بالدرجن

only 2 Post root
PN

COMA

DEFINITION

- A state of profound **UNCONSCIOUSNESS** from which the patient cannot be aroused, even by powerful stimuli.
- According to Glasgow coma scale (GCS): Complete loss of consciousness with:

- No eye opening.
- No verbal response.
- No motor response.

GCS = 3

- According to Glasgow coma scale (GCS):

General grading of the level of consciousness is recently done according to the patient's response to external stimuli, using **3 criteria:**

- Eye opening: 4 grades.
- Verbal response: 5 grades.
- Motor response: 6 grades.

Eye Opening	Verbal Response	Motor Response
Spontaneous 4	Oriented 5	Obeys orders 6
In response to speech 3	Confused 4	Localizes to pain 5
In response to pain 2	Words, no sentences 3	Withdraws to pain 4
None 1	Sounds, no words 2	Flexes to pain 3
	None 1	Extends to pain 2
		None 1

تدريس على حاجب - nipple

لا يفتح عينه بالرغم من كل حاجب

ليقول جمل مناسبة

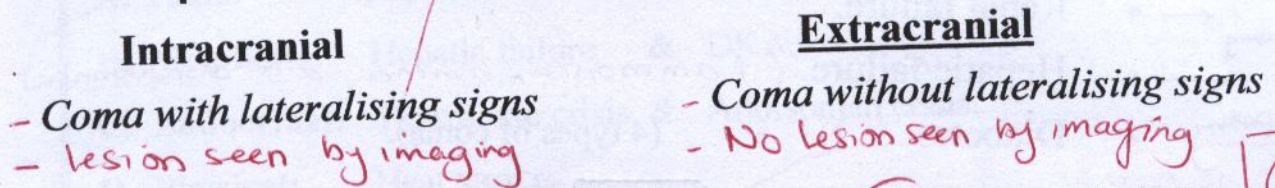
حجب يده في المكان لسحب يده

عقلانية لا تشبه decerebrate response

عقلانية التشبه decorticate response

WhiteKnightLove

ETIOLOGY



1. INTRACRANIAL CAUSES

- Traumatic: Head injury, e.g. cerebral concussion, contusion, laceration.
 - Inflammatory: Encephalitis, meningitis, brain abscess.
 - Neoplastic: Brain tumour.
 - Vascular: Hemorrhage (Cerebral & subarachnoid), thrombosis, embolism, Hypertensive encephalopathy.
 - Epilepsy.
- Handwritten notes:* "Local causes in the brain" (circled), "infarction", "Hge 20-25 min".

2. EXTRACRANIAL CAUSES

A. Toxic:

1. Aspirin (salicylate) poisoning:

- **Fever**, bleeding tendency, hyperventilation, vomiting.
- Handwritten notes:* "antiplatelets", "salicylic acid", "pH acidosis → ↑ RC".

2. Alcohol intoxication:

- Characteristic mouth odour, flushed face & conjunctiva, ↑ blood alcohol.
- Handwritten notes:* "VD", "headache", "والأفعل في الغيوة", "سوء المزاج".

3. Barbiturate poisoning:

- Respiratory depression, circulatory failure, ↑ blood barbiturates.
- Handwritten notes:* "↓ RC", "↓ VMC".

4. Belladonna (atropine) poisoning:

- **Fever**, hot flushed dry skin,
- Handwritten notes:* "Bilat. dilated pupils", "↑ RC".

5. Carbon monoxide poisoning:

- Cherry red skin.

6. Morphine (opiate) poisoning:

- Pinpoint pupils, respiratory depression, hypothermia, bradycardia.
- Handwritten notes:* "direct effect", "↓ RC", "↓ HRC", "Heat Regulatory Center", "Peripherally", "as parasympathetic", "upper hand is sympathetic".

B. Metabolic:

- Respiratory failure.
- Renal failure.
- Hepatic failure. (↑ ammonia, amines, a.a. disturbance)
- Diabetes (4 types of coma).

Uremic encephalopathy

Hepatic encephalopathy

hypoxic
hypercapnia

both cause R.F

Fever

C. Endocrinal:

- PITUITARY: Hypopituitarism.
- THYROID: Hypothyroidism & thyrotoxic crisis.
- ADRENAL: Addisonian crisis.

Myxedema Coma

Fever

D. Physical:

- Heat stroke.
- Hypothermia. → it's most common cause is Myxedema

Fever

E. Cardiac auses:

- Cardiac arrest.
- Cardiac arrhythmias. → severe ↓ COP → syncope wif ↑↑ time

irreversible brain damage

F. Infections:

Fever

Intra cranial
extra cranial

- Meningitis.
- Malaria, especially the Cerebral type.
- Septicemia & fulminant infections.
- Status typhosus.

its target organ is intestine

its target organ RBCs & hepatocytes

لحم يبيطوا في الدم والمخ

WhiteKnightLove

سؤال وإنما في واحدة كذا شرح

CAUSES OF FEBRILE COMA

- A. Toxic: Aspirin & Belladonna poisoning.
- B. Metabolic: Hepatic failure & DKA.
- C. Endocrinal: Thyrotoxic crisis & Addisonian crisis.
- D. Physical: Heat stroke.
- E. Fevers: ALL causes of fevers causing coma.
- F. Vascular: Pontine hemorrhage & subarachnoid hemorrhage.
- I. Any type of coma complicated by secondary infection, e.g. hypostatic pneumonia.

irritation of HRC

retained secretions
& may lose cough reflex

CLINICAL APPROACH TO COMA

INITIAL GENERAL MEASURES

- Ensure free airway passages. ETT → 1 - O₂
2 - secretion
3 - mechanical ventilation
- Ensure enough blood to the brain (maintain adequate BP).

أعرف سبب
Coma
as it is
H of cause

HISTORY TAKING

مناقشة

examples

1. Onset:

- Sudden: Cerebral hemorrhage
- Gradual: brain tumour.

2. Head injury.

3. Convulsions:

Epilepsy, brain tumours.

4. Drug intake:

Drug poisoning.

Hge in epileptic
at start

5. Excessive exposure to the sun: Heat stroke.

6- Diabetic or Hypertensive

GENERAL EXAMINATION

A) VITAL SIGNS

1. Temperature:

- Hyperthermia: Causes of febrile coma.
- Hypothermia: Hypothyroidism, Morphine poisoning.
↓ HRC

2. Pulse:

- Tachycardia: Thyrotoxic crisis.
- Bradycardia: Hypothyroidism, Morphine poisoning.
vagal stimulation → HB
adam's stroke

3. Blood pressure:

- High: Hypertensive encephalopathy.
- Low: Addisonian crisis.

4. Respiration:

Kausmaull's

- Deep, rapid: DKA, Renal failure.
- ~~Deep~~ slow: Barbiturate poisoning, Morphine poisoning.
↓

B) MOUTH ODOUR

- 1. Acetone odour: DKA.
- 2. Alcohol odour: Alcohol intoxication.
- 3. Urineferous odour: Renal failure.
- 4. Fetor hepaticus: Hepatic failure.

C) SKIN EXAMINATION

- 1. Dry skin: DKA, Belladonna poisoning.
- 2. Moist skin: Hypoglycemic coma.
- 3. Cherry red skin: Carbon monoxide poisoning.
- 4. Needle marks: → Drug addiction.

سکسکت

هرش

5 scratch

→ Hepatic failure
Renal failure
DKA

158

→ Catecholamine
→ Sympathetic activity
→ Sweating
White Knight Love

CNS EXAMINATION

1. Signs of lateralisation: denote an intracranial cause for the coma.

SIGNS OF LATERALIZATION

- EYES: Unequal pupils, Deviation of the eyes to one side.
- FACE: Facial asymmetry. *to healthy side*
- MOTOR: Jacksonian fits, (Unilateral paralysis or paresis.)
لا تكبر الا لو كان الحيات يقدر
- DEEP REFLEX & TONE: Asymmetry. *يستاءن على*
- SUPERFICIAL REFLEX: Unilateral positive Babinski (not in deep coma)

2. Signs of meningeal irritation: Meningitis, SAH.

*Kernig
Lasegue
Brudzinski* → *tue in* ← *meningitis (coma)
SAH
Scintica (no coma)*

3. Pupils:

- Dilated fixed:
 - Unilateral: 3rd nerve compression, as in uncal herniation.
 - Bilateral: Belladonna poisoning.
- Constricted:
 - Bilateral: Pontine hemorrhage & Morphine poisoning (pin point).

1st is irritative then paries on opposite side of lesion

4. Fundus examination: papilloedema in cases of increased ICT.

INVESTIGATIONS *(The definitive diagnosis)*

A) IMAGING

1. Plain x-ray skull:

- Features of ICT: brain tumours.
- Fractures: trauma.

2. MRI & CT scan: **"The most important investigations"**

- They are very accurate in diagnosing intracranial lesions.

*Clinical Q
PPP*

*Toxic - Morphine
- Pontine Hge
↓
damage of sympathetic fibers.*

if normal CT & MRI
على الخطوات دي تساعد

B) LABORATORY INVESTIGATIONS

الفقر

1. Blood examination:

examples

- CBC: leucocytosis in bacterial infections. ^{shift to lt}
- Blood film: malaria.
- Blood levels of: ^{DKA} glucose, ^{RF} urea & creatinine, ^{Hepatic} bilirubin.
- Blood gases: respiratory failure. ^{hypoxia} ^{hypercapnia} ^{ear ear}
- Toxicological studies: drug poisoning.

Types
II I

2. Urine examination:

- Glucose. → hyperosmolar non ketotic
- Acetone. → DKA
- Albumin. → RF

لم ياف
أخذتولين
أعني على زنا راع
عنه راع

3. CSF examination:

- Bloody: SAH.
- Purulent: Meningitis.

TREATMENT

أشرح كامل

1. Care for the comatosed (as in hemiplegia).

2. Treatment of the cause. eg: DKA

أشرح كامل
endocrinal
الا لرسؤال قصير اكتب فقط

1 Insulin

2 fluids IV

أشرح
Myasthenia me-
PN, Myo +
Myasthenia
neurologic brain
disease

Epilepsy

neurotransmitters

excitatory

Inhibitory

balance

هنا فيه خلل في هذا الميزان لصالح ان
excitatory ↑ ↓ inhibitory ↓

Excitation of Brain cells

كهرباء زيادة بالبدي

epileptogenic focus = hyper excitable neurones

تستغل زيادة عنه للزفرة

motor actions ↑

motor area 4

وال C/P على حسب مكانه فيه

Parasthesia

sensory area 1, 2, 3

Jacksonian fit may be sensory or motor

الخطبة في
كهرباء المخ

أي ما قلب
فيه كهرباء ولما
تلتخط بعمل
anhythmia
يصرف النظر عنه
سبب الخطبة فيه

* مريض نوع *arrhythmias* خالٍ من تشخيص بحاجة غير ECG
عالمش وجود

* وكذلك مريض نوع *epilepsy* خالٍ من تشخيص بحاجة غير EEG

الشراة باعة
epilepsy

oral

EEG is the most important investigation في النوع والأخيرة

introduction about functions of brain

Normally

Focus ← مركز يكون في جزء من المخ الذي الخ
سبب السبب بقاء

if disturbed "hyperexcitable"

1. Somatic functions (Bilaterally)

= unilateral on opposite side (due to crossing)

Centers in Cortex

1. Motor (area 4)

Motor Jacksonian fits

2. sensory (area 1, 2, 3)

Sensory

burning numbness tingling

3. special senses

olfaction (uncus)

olfactory

hallucination

vision (area 17)

visual

hallucination

auditory (41, 42)

auditory

hallucination

2. Autonomic functions

من أول حاجب ففكر في من (منع) كمنع في حسابك

Centers in Hypothalamus

1. Motor autonomic

eg: GIT motility

Abdominal colic, diarrhea, vomiting

Bladder

Urine incontinence

heart

Tachycardia

Skin { sympathetic parasympathetic }

Sweating

2. Sensory autonomic

حساسية

nausea, abdominal distension

dyspnea, palpitation

3. Psychological functions (behaviour)

Behavioral changes

Centers

1. Prefrontal area

Hallucination

تصريفات
مفكوسة

2. Limbic system

Temporal lobe epilepsy

temporal lobe epilepsy

4. Consciousness Level

حضر لا يمكن oral من أنواع

Centers in 1. RAS 2. cortex

epilepsy →

White Knight Love

* aura: precedes the epilepsy:

سبب رضة ← electric discharge
كثرة وتيرة وقت قليلة
وهي من جنس ال attack

Mechanism of epilepsy → 2ry causes (General causes)

one or more of following mechanisms

دافع

1- Cortical irritation & stimulation of Brain cells and/or facilitation of neurotransmission

لا يكتب في النظرى
لكنه عشان لازم

through inhibition of inhibitory transmitters (GABA)

- eg: Fever
- eg: Electrolytes

لذلك العلاج هو دواء
يهدئ الخلايا ويزود GABA

2- Brain hypoxia

- eg: Fallots Tetralogy: infundibulum → Cyanotic spells
- eg: Adam stroke's attack: Complete Heart Block

الدم يقل
لا يدخل

Focus [idioventricular rhythm] →

لكنه ال focus
ده وقف
مفيع

3- Toxic effect "Toxins pass BBB from Blood to brain"

- eg: Renal failure
- eg: Liver failure
- eg: Alcohol

MSOF → (واحد منهم هو الخ)

ضغط نبض
مانع على ترويض
عنه تشنجات

4- Nutritional deficiency → electrical instability

- eg: ↓ B₁ (vitamins)

EPILEPSY

1 DEFINITION

- It comes from the GREEK name "**Epilepsia**" which means "**taking hold of** or **seizing**".
- It is a disorder characterized by:
 - 1 spontaneous tendency for recurrent **seizures**.
 - 2 free between attacks

SEIZURES

Recurrent transient attacks of:

somatic, **psychic**, or,

autonomic clinical features.

Represent: (reflects)

clinical features of abnormally hyperexcitable cortical neurons.

Result from:

paroxysmal and **excessive** electrical neuronal discharges.

ASSOCIATED WITH:

EEG changes & may be disturbance of consciousness.

2 ETIOLOGY

1. Idiopathic epilepsy

- It is the commonest cause.
- It may be associated with **positive family history** in some cases.
- It starts in the 1st & 2nd decades in the form of:
 - Grand mal epilepsy.
 - Petit mal epilepsy.
 - Myoclonic epilepsy.
 - Atonic seizures.

no cause can be detected (65%) → **HCCQ**

in 30% **HCCQ**

2. Secondary epilepsy

A. Local causes in the brain:

1. Congenital: cerebral palsy.
2. Traumatic: cerebral contusion or laceration.
3. Inflammatory: encephalitis, meningitis, brain abscess.
4. Neoplastic: brain tumours.
5. Degenerative: presenile dementia.
6. Vascular: stroke (especially hemorrhagic), hypertensive encephalopathy.

B. General causes with secondary effects on the brain:

1. Toxic: Alcohol, cocaine, lead, Botulism, tetanus, CO, cyanide.
2. Iatrogenic: Lidocaine, INH, Ambilhar, Amphetamine, Aminophylline.
3. Metabolic: ↑ glucose & ↓ glucose, ↑ Ca & ↓ Ca, - ↑ Na & ↓ Na.
4. Endocrinal: Hypoparathyroidism → ↓ Ca, - Hyperthyroid crisis.
5. Organ failure: Hepatic failure, - Renal failure.
6. Heart disease: Adam's Stoke's attacks, - Fallot's tetralogy.
7. Nutritional: Pellagra, B₃, - Vitamin B₆ deficiency.
8. Physical: High fevers, - Heat stroke.
9. HYSTERICAL. → psychosomatic

TOXIC

Organ failure

Iatrogenic

metabolic

endocrinal

physical

Toxic effect

Cerebral irritation effect

Heart disease

+ Respiratory failure

↓ O₂ ↑ CO₂

hypoxic TOXIC

hypoxic effect

White Knight Love

3

CLINICAL PICTURE

1. GENERALISED SEIZURES

"Excessive electrical discharges from cortical neurons in **BOTH** hemispheres simultaneously"

I. Grand Mal Epilepsy:

"attacks of tonic-clonic convulsions"

1. Pre-ictal stage (seconds) (aura)

- 1 - It is a warning sign of a coming attack.
- 2 - It may be: (brief electrical discharge)

- Somatic: Myoclonus, Parasthesias. (burning tingling numbness)
- Psychic: Hallucinations.
- Autonomic: Tachycardia, Sweating, Colic

2. Ictal stage (seizure)

- 1 - Sudden loss of consciousness: for seconds to minutes.
- 2 - Tonic phase (few seconds) = sustained contraction

- The UL & LL: are extended.
- The HEAD: is retracted to one side & the eye balls rolled up.
- The BACK: High arched (opisthotonus)
- The JAWS: are firmly clenched, with biting of the TONGUE.
- CYANOSIS: due to impaired respiration.
- There may be incontinence of urine.

- 3 - Clonic phase (few minutes) = Contraction then Relax

- The UL & LL: contract & relax repeatedly & rapidly.
- The HEAD: jerks forcibly.
- may be incontinence of urine

3. Post-ictal stage (sequelae)

- It may be:

- 1 • Somatic: Todd's paralysis (< 24 hours, due to neuronal exhaustion).
- Psychic: Confusion.
- Autonomic: Vomiting.

Drug of choice: Carbamazepine (Tegretol) or Phenytoin (Epanutin)

II. Petit Mal Epilepsy: "attacks of loss of consciousness" "Absence"

1. It starts in childhood & improves at puberty & usually disappears at the age of 20.
2. It is NOT PRECEDED by aura & NOT FOLLOWED by sequelae.
3. It is usually PRECIPITATED by: hyperventilation or photic stimulation.
4. It is characterized by: sudden loss of consciousness of short duration (few seconds).
5. It may be associated with:

- High frequency (50 attacks / day) = **picnolepsy**
- Falling to the ground without warning.
- Jerky movements of the head & UL (myoclonic petit mal).

Drug of choice: Valproate (Depakine) or Succinimide (Zarontin)

127

General causes of 2ry idiopathic

لو اسباب غير معروفة
status epilepticus
ممكن عيوت

jerky movement
contractions followed by Relaxation

Never exceed 30 min
if exceeded => status epilepticus

لذلك لا زلزال
فم مفتوح
قسطه جوة بقاء
عشان ميعطش بالاسان
R.F. تحوت

urine incontinence
clonic مع 35
منظري 12 من
relax

تايم منك لما فاقه
due to Brain hypoxia
due to Resp Muscle strain

الفاروق
تباع 4
انه هنارة
unilat.
Bilat

اهم دواعي

associated
loss of body tone

staring
gazing

اشهر الاسباب واحدة في جهة الارباعي
ما ساه غير حاجه اجابتي حاجات قبله attack عند الشللة
White Knight Love

myoclonic 3-times ← aura of grand mal
petit mal
لوحده

III. Myoclonic Seizures: "attacks of involuntary clonic movements"

Onset: sudden
Offset: sudden

- It is characterized by: sudden, jerky, shock-like INVOLUNTARY muscle contraction.
 - The jerks are bilateral contractions, mainly of the shoulders and arms.
 - However, some patients report jerking in the lower limbs, trunk, or head.
- It may be of 2 types:
 - Simple: - Occurs singly (no loss of consciousness).
 - As a part of: - Grand mal epilepsy (aura). - Petit mal epilepsy.

Drug of choice: Valproate (Depakine) or Clonazepam (Rivotril)

IV. Atonic seizures: V.V.V. rare

- Transient attacks of brief loss of postural tone, often resulting in falls and injuries.

2. PARTIAL SEIZURES

"Excessive electrical discharges from cortical neurons in a certain area in ONE hemisphere"

A. Simple seizures:

"No disturbance in consciousness"

- The CP depends on the site of the hyperexcitable neurones in the cerebral cortex, whether in: "Motor area or Sensory areas".

1. Motor fits:

- Focal fits:
- Motor jacksonian fits:

2. General Sensory fits:

- Focal fits:
- Sensory jacksonian fits:

3. Special Sensory fits:

- Visual hallucinations:
- Auditory hallucinations:
- Olfactory hallucinations:

movement of part of a limb or the whole limb.
movement of one side of the body (see before).

parasthesia of part of a limb or the whole limb.
parasthesia of one side of the body (see before).

irritation of the visual sensory area.
irritation of the auditory sensory area.
irritation of the uncus.

B. Complex seizures:

"disturbance in consciousness"

SITE: The hyperexcitable neurons are in the Temporal lobe
DURATION: The seizure lasts few seconds to few minutes.

The seizure starts with Aura, followed by Absence, Automatism, Amnesia:

1. **Aura:** Olfactory hallucinations, Déjà-vu phenomenon, Sensation of fear.
2. **Absence:** Absent patient with staring eyes (with no response to conversation).
3. **Automatism:** Involuntary Purposeless acts: motor (eg, lip smacking, chewing,) or verbal.
4. **Amnesia:** No recalling of the seizure.

no motor auras
no general sense

Smelling recent memory behaviour
White Knight Love

3. PARTIAL SEIZURES → GENERALISED SEIZURES

"Partial seizures may spread to involve the whole brain → secondarily generalised seizures".

Psychogenic Epilepsy

Hysterical

- The cause is psychological & there is no organic lesion.
- ↑ • It usually occurs in the presence of people.
- ↓ • It never occurs during sleep.
- The patient never hurts himself: e.g. there is no tongue biting.
- There is no incontinence of urine.
- The EEG is normal.

حالة غير عضوية
organic

4

PRECIPITATING FACTORS

- Missed AED → stimulates GABA [فانغ بروج-شغل]
- Menses.
- Alkalosis. $\uparrow \text{pH} \rightarrow \downarrow$ threshold of brain cells stimulation
- Alcohol use & Drug abuse (e.g. cocaine). (excitatory)
- Stimulation by photons & Hyperventilation.
- Sleep deprivation & Stress & sudden withdrawal of antiepileptic drugs.

Causes disturbance of balance between Excitatory & inhibitory in favor of excitatory

لأنه يسبب اختلال التوازن بين المثبط والمثير لصالح المثبط

أثناء العلاج
عاز الحماض
يكون على حافة
acidotic
عشاق يرفع
threshold

5

INVESTIGATIONS

1. EEG:

- It is the most specific test for epilepsy because it records the electrical activity of the brain.
- It shows specific pattern: "Epilepsy waves".

2. LOCAL INVESTIGATIONS:

"CT & MRI of the brain"

- To identify or exclude a LOCAL CAUSE of seizures in the brain.

eg: Hge or tumor

3. GENERAL INVESTIGATIONS: "Laboratory investigations"

- To search for a GENERAL CAUSE of seizures, e.g. blood glucose.

$\downarrow \text{Ca}$

6

TREATMENT

A. General Measures:

1. Moderation of the patient's physical activity.
2. Avoid the precipitating factors (Alcohol, hyperventilation, photic stimulation.....).
3. A ketogenic diet is encouraged because it will induce acidosis:
- Acidosis is beneficial as it raises the threshold of stimulation of the brain cells.

B. Specific Treatment:

1. Treatment of the cause in secondary epilepsy. eg Control
2. Anti-epileptic drugs:
 - a) Always start with one drug, then add another drug if there is no response.
 - b) Always stop the drugs ONLY if:
 - The patient stays free of symptoms for at least 2 years.
 - The patient has a normal EEG.
3. Side effects of Anti-epileptic drugs:
 - 1. Skin rash.
 - 2. Bone marrow depression. (aplastic anemia)
 - 3. Ataxia. toxic on cerebellum (years)
 - 4. Drowsiness

ANTI-EPILEPTIC DRUGS

Drug	Dose	Best indicated
1. Barbiturates: Luminal (Phenobarbitone)	100-600 mg / day	- Broad spectrum. - Not for petit mal.
2. Hydantoin: = phenytoin Epanutin	100-600 mg / day	- Grand mal. - Motor Jacksonian fits.
3. Carbamazepine: Tegretol	200-600 mg / day	- Grand mal. - Motor Jacksonian fits. - Complex seizures. - Not for petit mal.
4. Clonazepam: Rivotril	2 - 6 mg / day	- Myoclonic. - Grand mal.
5. Valproate: Depakine	500-1500 mg / day	- Broad spectrum.
6. Succinamide: Zarontin	500-1000 mg / day	- Petit mal.

NEW ANTI-EPILEPTIC DRUGS

- These drugs are new drugs that may be used in resistant seizures.

1. Lamotrigine: 200 - 400 mg / day.
2. Felbamate: 400 - 800 mg / day.
3. Gabapentin: 600 - 1200 mg / day.

gradually stop drugs
- anti-epileptic
- anticoagulant
- cortisol



STATUS EPILEPTICUS

DEFINITION

- A medical emergency where there are:
 1. Repeated attacks of **generalized convulsions**, with **lack of recovery of consciousness**, **OR**,
 2. **Persistent attack of seizure lasting for at least 30 minutes.**
- If the convulsions are not stopped rapidly, **coma deepens** & death may occur due to: **heart failure** or **respiratory failure** or **brain damage** or **hyperpyrexia**.
- The most common causes are: **sudden withdrawal of anti-epileptic drugs** & **stroke**.

TREATMENT

A. General Measures:

1. Take care of: **"ABC"**
 - Place the patient on the **ground**, to guard against falling from bed.
 - Mouth gag & O2 inhalation** (endo-tracheal intubation may be needed).
 - Record the **vital signs** regularly.
 - RR → R.F.
 - HR, BP → H.F.
 - Temp → Fever
2. Take a sample of:
 - Venous blood: for the level of: **anti-epileptic drugs, alcohol.**
 - Arterial blood: for the level of: **pH, pO₂, pCO₂, HCO₃.**

3. Give cerebral dehydrating measures: e.g. **Lasix, conc. Mannitol, Dexamethazone.**

B. Specific Treatment:

- **Epanutin with Valium (or Rivotril) are given immediately:**

1. **EPANUTIN** (Phenytoin): 15 mg / Kg slow infusion.
2. **VALIUM** (Diazepam): 5 mg slowly IV, to be repeated after 5 minutes if seizures recur: maximum dose: 20 mg.

OR:

1. **RIVOTRIL** (Clonazepam): 2 mg slowly IV, to be repeated after 5 minutes if seizures recur: maximum dose: 6 mg.

- **If seizures persist after 20 min. of Epanutin & Valium:**

3. **PHENOBARBITONE:** 200 mg infusion.

- **In resistant cases:**

4. **GENERAL ANAESTHESIA:** may be used.

- تستخدم في هذا الكتاب
- uses
1. stroke (↓ edema)
 2. Bell's Palsy (canal)
 3. GBS
 4. Myasthenia
 5. MS
 6. Meningitis

Muscle relaxation
Life saving

2ry myopathy